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Table of Contents.

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ORIGINAL ARTICLES— Page.
The Rh Factor: Laboratory Aspects, by Rachel Jakobowicz and Lucy M. Bryce 477
Importance of the Rh Factor in Obstetrics, by Vera Krieger, D.Sc. 480
The Rh Factor: Ethnological Aspects, by R. T. Simmons *et alii* 483
A Time-Saving Mastoidectomy Dressing, by J. R. Hutcheon, M.R.C.S., L.R.C.P., D.L.O. 485

REPORTS OF CASES—
Cancer of the Female Urethra, by W. G. Cuscaden, M.D., F.R.C.S., F.R.A.C.S. 487

REVIEWS—
Medicine and Sociology 490

LEADING ARTICLES—
Carriers and Enteric Fever 491

CURRENT COMMENT—
Peritoneoscopy 492
Acute Generalized Miliary Tuberculosis 493

ABSTRACTS FROM MEDICAL LITERATURE— Page.
Physiology 494
Biochemistry 495
Medicine 495

BRITISH MEDICAL ASSOCIATION NEWS—
Medico-Political 496

MEDICAL SOCIETIES—
Melbourne Pædiatric Society 496

NAVAL, MILITARY AND AIR FORCE—
Appointments 499
Casualties 500

NOTICE 500

NOMINATIONS AND ELECTIONS 500

OBITUARY—
John Grantley Vincent Shelton 500

DIARY FOR THE MONTH 500

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 500

EDITORIAL NOTICES 500

THE RH FACTOR: LABORATORY ASPECTS.[1]

By RACHEL JAKOBOWICZ AND LUCY M. BRYCE,
From the Walter and Eliza Hall Institute of Research and the Red Cross Blood Transfusion Service (Victorian Division).

IT was considered that a short survey of the nature and relationships of the blood group factors in general might make the symposium to be presented tonight more intelligible to those members of this society who are not immediately concerned with work in this field.

As most of you probably know, Landsteiner discovered in 1900 that human red cells contained two agglutinable substances which he called A and B. Elaboration of this work showed that all human beings could be classified into four groups, by virtue of the fact that any individual's red cells possessed one or other or both or neither of these substances. Later work showed that substances of similar specificity were also present in other cells and body fluids, including urine and saliva, in a majority of persons. These persons are, in this connexion, called "secretors". The minority, in whose urine and saliva the A and B substances cannot be demonstrated, are called "non-secretors".

The serum of human blood normally contains reciprocal agglutinins reactive with the A and B factors. For example, the blood of a person of group A contains anti-B (sometimes referred to as β) agglutinins, which when mixed with group B cells, either *in vivo* or *in vitro*, cause these cells to become clumped together or agglutinated. These facts are of great practical importance in connexion with blood transfusion. For example, if blood of a group B donor was to be given to a person whose blood belonged to group A, and whose serum therefore would contain anti-B agglutinins, the introduced cells would be agglutinated in the recipient's blood vessels. Such reactions, which are likely to cause adverse symptoms or even the death of the recipient, can, of course, be avoided by using donors of suitable group.

Occasionally, however, even transfusions of blood from donors belonging to the same group as the recipient have been followed by so-called "intra-group" reactions with symptoms resembling those of "inter-group incompatibility".

From time to time since Landsteiner's original work, other agglutinable substances in red cells in addition to the A and B factors have been identified. These other factors have been called M and N, P *et cetera*. After their identification it was logical to try to ascertain whether they were the cause of the hitherto unexplained intra-group transfusion reactions. Human serum, however, does not normally possess reciprocal agglutinins for these factors as it does for A and B, nor (with occasional rare exceptions) does their introduction into the body stimulate the production of antibodies in human serum. Furthermore, in cases in which adequate investigation has been possible, no evidence has been obtained from tests *in vitro* (again with rare exceptions) that they do play any part in such reactions.

The latest red cell agglutinogen to be described is the subject of our papers tonight—the Rh factor. It owes its rather mystifying name to the fact that it was first identified by Landsteiner and Wiener in 1940 in the course of a study of inter-species relationships of such factors, during which they injected red cells of Rhesus monkeys into rabbits. The rabbits produced antibodies against these foreign cells, and it was subsequently found that their serum agglutinated, not only the Rhesus cells, but also those of some 85% of human beings. Those whose cells were thus agglutinated were said to possess the Rh factor (Rh being short for Rhesus), or alternatively, were called "Rh-positive". Those whose cells were not agglutinated were called "Rh-negative".

[1] Part of a symposium on the Rh factor held by the Victorian Society for Pathology and Experimental Medicine on December 13, 1943, at Melbourne.

Like M and N, P *et cetera*, the Rh factor is present chiefly in the red cells, and there is normally no reciprocal agglutinin in Rh-negative serum. Unlike M, N and P, however, the Rh factor does, in a not negligible proportion of cases, stimulate the production of antibodies if it is introduced into Rh-negative blood. It may be introduced in the following ways: (i) by transfusion, (ii) by immunization of the mother by her fœtus. In both types of case, antibodies may be formed which will agglutinate Rh-positive cells. If, therefore, a person with Rh-negative blood, who has previously been given Rh-positive blood and has reacted to it by producing anti-Rh agglutinins, is given further transfusions of Rh-positive blood, a post-transfusion reaction, sometimes very severe, may occur. Similarly, if a woman whose blood is Rh-negative has a child who has inherited the Rh factor from its father, the Rh-positive red cells of the fœtus may stimulate the formation of Rh antibodies in the mother's serum. Should such a woman need a blood transfusion during pregnancy or subsequently as long as her antibodies persist, she may suffer a transfusion reaction if given Rh-positive blood. Furthermore, her antibodies may pass back across the placenta and adversely affect her fœtus whose blood is Rh-positive, giving rise to conditions such as *erythroblastosis fœtalis*. The clinical implications of such interactions are being discussed by Dr. Vera Krieger, so I shall not dwell on them any further.

Technique of Testing for the Rh Factor.

Landsteiner and Wiener and their associates in America and leading English workers all recommend a mixture in tubes of weak suspensions (1% to 2%) of the cells to be tested with appropriate dilutions of serum known to contain Rh antibodies, or conversely, a mixture of serum suspected of containing anti-Rh agglutinins with suspensions of known Rh-positive and Rh-negative cells. This is still the method of choice for detection of agglutinins in serum. However, using a technique similar to that widely employed for routine blood grouping, we have found that a mixture of cells and serum on slides or tiles may be used for testing unknown cells, provided anti-Rh sera of sufficient potency are available. The slide or tile method is, of course, economical of time and material when large-scale surveys are made.

An important point is that usually the Rh agglutination is most active at 37° C., and is therefore sometimes referred to as "warm agglutination". In this it differs from the agglutination concerned with the ABO system, the M and N factors, and most other red cell agglutination reactions, which are either independent of the temperature at which the test is made (within the range from about 5° to 37° C.) or are noticeably stronger at the lower end of this range.

Another point of importance is that Rh agglutination seems to be the result of a much less firm union of cells than are the other types of agglutination mentioned above. For example, in routine blood grouping, weak reactions can usually be accelerated and intensified by continued agitation of tube, slide or tile; but if this procedure is adopted when one is testing for the Rh factor, the agglutinate is likely to be broken up and rendered unrecognizable. We therefore examine the sediment, by the "pattern" of which the presence or absence of Rh agglutination is determined.

Incidence of the Rh Factor in Red Cross Donors.

Dr. Jakobowicz's and my immediate concern in connexion with the Rh factor was to provide donors with Rh-negative blood for the Red Cross Blood Transfusion Service, and our initial problem was to obtain suitable anti-Rh serum in sufficient quantities.

The first serum obtained was from a volunteer for the Red Cross Service, Mrs. Chl. Her red cells belong to group A; but Dr. Jakobowicz found that her serum agglutinated the cells of a large number, but not all, of samples of cells of groups A and O. We were fortunate in that at that time the Commonwealth Serum Laboratories were in possession of small samples of human and guinea-pig anti-Rh serum, which had been sent to them by Dr. Landsteiner. It was then ascertained by Mr. Simmons and Mr. Graydon that the agglutinins in the serum of our donor corresponded to those in their known anti-Rh sera. It was also ascertained that some eight months before she volunteered as a donor, Mrs. Chl. had given birth to a baby suffering from *erythroblastosis fœtalis*.

Mrs. Chl.'s serum contains potent anti-Rh agglutinins, and she has continued to act as our main source of supply, having up to the present time given a pint of blood on three occasions at intervals of three months. Her serum can be used for testing cells of group A or group O; but it is necessary to remove her anti-B agglutinins before it can be used for testing cells of group B or group AB. This has been done by absorption with group B Rh-negative red cells, or with saliva, either unmodified or dried, from a group B "secretor".

Mrs. Chl.'s serum was "processed" at the Commonwealth Serum Laboratories, and as soon as sufficient was available, the Rh classification of our donors was commenced. During the earlier stages of this work, when a comparison of various methods was being made, Mr. Simmons and Mr. Graydon collaborated by testing, in parallel with our tests, the cells of some 2,000 donors whose blood had been sent to the Commonwealth Serum Laboratories for the preparation of pooled serum for transfusion. Since then Dr. Jakobowicz, assisted by Dr. Karoly, has continued to classify our donors. Table I shows the results obtained with 5,568 samples of blood tested with Mrs. Chl.'s serum. It will be seen that the proportion of persons with Rh-negative blood is higher than that given in most reports from overseas (17·7% as against 15%). As will be indicated later, this difference would seem to be best explained by postulating the absence of at least one partial agglutinin from Mrs. Chl.'s serum.

TABLE I.

Results of Testing 5,568 Samples of Red Cells with Anti-Rh Serum Chl.

Blood Group.	Number Tested.	Rh-Negative Red Cells.	
		Number.	Percentage.
O	2,550	435	17·06
A_1	1,557 } 2,240[1]	292 } 424[1]	18·75 } 18·93[1]
A_2	461 }	90 }	19·52 }
B	582	98	16·85
AB	196	29	14·79
Totals ..	5,568	986	17·71

[1] Total group A including some blood samples not tested for sub-groups.

We have subsequently obtained several other sera, some from Dr. Krieger at the Women's Hospital, and some through the collaboration of medical practitioners, among whom we particularly wish to thank Dr. Julian Smith and Dr. Ella Macknight. With these sera we have retested all samples of cells which did not react with Mrs. Chl.'s serum, so that all those now classified as Rh-negative have been tested with three to five sera in addition to that of Mrs. Chl. These retests have reduced the incidence of Rh-negative persons to 16·8%.

Comment.

These investigations indicate a difference between the Rh reaction and that concerned with the ABO system. Although it has been shown that anti-A and anti-B agglutinins are composite substances, in that they contain fractions having different affinities for animal cells or for the group-specific receptor substances in human saliva or serum, they nevertheless react with all human A or B red cells, provided the technique used is adequate. On the other hand, the Rh agglutinin in one person's serum may fail to agglutinate red cells which are agglutinated by the Rh agglutinin in another person's serum. Table II shows the results obtained with 200 unselected samples of group O cells tested with six individual sera (Chl., P1, Gl, Mi, P2 and Si).

An analysis of these results shows that the majority of cells (142) reacted with all six sera, 18 reacted with four sera (Chl., Gl, P1 and Mi), two reacted with another

TABLE II.

Agglutination of 200 Unselected Samples of Red Cells by Six Human Anti-Rh Sera, and Comparison with Results Obtained by Levine.

Sera.	Postulated Partial Agglutinins Present.	Number of Cell Samples Agglutinated.				Total Rh-positive.	Total Rh-negative.	Percentage Rh-negative.
		142	18	2	1	163	37	18·5
Sera tested by R.J. and L.B.:								
Group I. Chl and Gl ..	ρ1 ρ3 ρ4	+	+	−	+	161	39	19·5
Group II. P1 and Mi ..	ρ1 ρ2 ρ3	+	+	+	−	162	38	19·0
Group III. P2 and Si ..	ρ2 ρ3	+	−	+	−	144	56	28·0
		Percentage of Cell Samples Agglutinated.				Percentage Rh-positive.	Percentage Rh-negative.	
		70	15	2		87	13	
Sera tested by Levine:								
Type 1	Rh 1	+	+	−			15	
Type 2	Rh 1 2	+	+	+			13	
Type 3	Rh 2	+	−	+			28	

combination of four sera (P1, Mi, P2 and Si), and one reacted with two sera only (Chl. and Gl). It will be seen, therefore, that in respect of the Rh reactions our six sera fall into three groups, and that the results we have obtained could be explained on the assumption that these three groups of serum contain different combinations of four partial agglutinins. It is of interest to note that the sera referred to as P1 and P2 were obtained from the same woman, P1 a fortnight after the birth of an erythroblastotic baby, and P2 some two months later. During this interval, not only had the anti-Rh titre fallen considerably, but it was found that P2 reacted with only 74·5% of the cell samples as compared with 82% classified as Rh-positive with P1 serum. P2 therefore most closely resembled serum Si, whereas P1 gave similar reactions to serum Mi. This change could be explained by postulating the disappearance of one of the partial agglutinins (ρ1) which was originally present in this woman's serum.

Levine describes three types of anti-Rh serum; he classifies them as anti-Rh 1 2 (giving an Rh-negative percentage of 13), anti-Rh 1 (giving an Rh-negative percentage of 15) and anti-Rh 2 (giving an Rh-negative percentage of 27). Our findings differ from his, in that none of the sera so far examined by us have agglutinated all cells tested as did his anti-Rh 1 2. There is, however, a general resemblance, in that both Levine's results and ours suggest the conception of the Rh agglutinin as a complex of partial agglutinins; but as our sera differ in the respects indicated above from Levine's R1 and R2, we have tentatively called our postulated partial agglutinins ρ1, ρ2, ρ3 and ρ4. According to this conception our group I (Chl. and Gl) contains partial agglutinins ρ1, ρ3 and ρ4, group II (P1 and Mi) contains ρ1, ρ2 and ρ3, and group III (P2 and Si) contains ρ2 and ρ3.

If we accept this interpretation of the nature of the Rh agglutinin, there seems no reason to stop at four partial agglutinins, and it is possible that the slightly higher incidence of Rh-negative cells in our series as compared with those of most other workers can be explained by the absence from any of the sera so far examined of additional

TABLE III.

Postulated Agglutinogen Patterns in Red Cells Suggested by Results of Absorption Tests.

Cell Samples.	Rh-Agglutination by Each Group of Sera.			Results of Absorption with Each Group of Sera.	Notes on Agglutination and Absorption Tests.	Cell Agglutinogen Type Postulated as a Result of Absorption Tests.
	Group I. (ρ1, ρ3, ρ4.)	Group II. (ρ1, ρ2, ρ3.)	Group III. (ρ2, ρ3.)			
1	−	+	+	Group I. Not tested. Group II. Not tested. Group III. Not tested.	Agglutination occurs only with sera containing ρ2. Therefore absorption not necessary.	R2
2	+	+	+	Group I. Poor. Group II. Moderate. Group III. Moderate.	Some absorption with all groups, but not good with any. Therefore probably due to one factor only (ρ3) common to all groups.	R3
3	+	+	+	Group I. Good. Group II. Moderate. Group III. Moderate.	Absorption with all groups, but best with Group I. Therefore probably due to ρ3 common to all groups together with ρ4 present in group I only.	R3, R4
4	+	−	−	Group I. Not tested. Group II. Not tested. Group III. Not tested.	Agglutination present with group I only. Therefore probably due to factor (ρ4) present only in this group.	R4
5	+	+	−	Group I. Moderate. Group II. Moderate. Group III. None.	Absorption present only with groups containing ρ1.	R1
6	+	+	+	Group I. Moderate. Group II. Good. Group III. Moderate.	Absorption with all groups, but best with group II. Results suggest that absorption with group I is due to ρ1, with group II to ρ1 and ρ2, and with group III to ρ2.	R1, R2

partial agglutinins. It is to be noted that the agglutinin pattern is independent of titre, since serum Chl. is very potent, being active at a dilution of 1 in 1,000 when tested by the "tube technique", whereas serum Gl with the same agglutinin complex will not react at any dilution greater than one in four.

It would seem that, corresponding with these partial agglutinins, there are also partial agglutinogens in the red cells, either singly or in combination, which can be demonstrated by absorption tests.

The number of absorption tests so far carried out has of necessity been small, as only limited supplies of some of the testing sera are available; but the results obtained, which are summarized in Table III, suggest that at least six varieties of cells can be differentiated. We do not know whether (even if it were genetically possible) these cell varieties represent actual subgroups comparable to the subgroups of the A antigen. The last-mentioned have been shown to be sometimes associated with reciprocal agglutinins, since blood which contains cells belonging to subgroup A_1 may contain anti-A_2 agglutinins in its serum and *vice versa*. So far we have not encountered analogous agglutinins in relation to the different varieties of the Rh agglutinogens. It is clear, however, that the varying degrees of agglutinability of red cells by anti-Rh sera is not merely a matter of varying "sensitivity", since a sample of cells which is only weakly agglutinated by one anti-Rh serum may react strongly with another anti-Rh serum, presumably because the agglutinin complex of the latter serum corresponds more closely with the particular agglutinogen complex of the cells in question than does that of the former serum.

This outline of some of the serological aspects of the Rh factor is necessarily brief, owing to the time limit for this paper; it is also incomplete, as we are following the suggestion made at a recent meeting of this society that work still in progress should be discussed. We hope, however, when further data have been collected, to report our results more fully.

Even at this stage, however, our data indicate the complexity of the Rh factor, and the problems to be faced by those attempting the classification, for practical purposes, of the blood of human subjects as Rh-positive or Rh-negative.

Perhaps the chief of these problems are the determination of the agglutinin pattern of anti-Rh sera, and their supply and processing in adequate amounts if found suitable for use in testing. Since the production of large amounts of immune serum in animals has not yet become a practical proposition, it would seem that the best method of dealing with these problems is active collaboration between (i) workers likely to encounter such serum in human beings, such as those associated with maternity hospitals, (ii) those having facilities for its collection and analysis, such as the Red Cross Blood Transfusion Service, and (iii) laboratories able to carry out their processing on a large scale, such as the Commonwealth Serum Laboratories. In Melbourne, we are fortunate in having such cooperation, and are developing the process of exchanging as far as possible samples of all useful serum, and we hope that from such coordinated work some useful results will be obtained.

IMPORTANCE OF THE RH FACTOR IN OBSTETRICS.[1]

By VERA KRIEGER, D.Sc.,
Melbourne.

As Dr. Bryce has already mentioned, about 15% of the population among white races has Rh-negative blood, the blood of the other 85% being Rh-positive. There is, however, a startling reversal of these figures in the group of women who have given birth to premature or full-term infants suffering from erythroblastosis. This term is used to cover a group of conditions of widely different clinical character, which have, however, certain features in common, namely: (i) the occurrence in the blood of numerous nucleated red cells (erythroblasts—hence the collective term erythroblastosis), and (ii) the continuation of the embryonal type of red cell formation outside the bone marrow in the various organs—for example, the liver, spleen, kidneys *et cetera*. No definite cause of this condition had been formulated until it was found that the blood of 90% of these mothers was Rh-negative and that of only 10% Rh-positive. Levine and his collaborators were the first to suggest the solution of the problem along the following lines.

The child of a mother with Rh-negative blood and a father with Rh-positive blood frequently has Rh-positive blood. The Rh substance in the baby's red blood corpuscles is thought to cause isoimmunization, with the production of Rh isoagglutinins in the serum of the mother. These antibodies may filter through the placenta into the fœtal circulation. The presence of these antibodies (i) in the serum of the mother and (ii) in the serum of the fœtus may be responsible for serious if not fatal consequences for each. Transfusion of Rh-positive blood into the mother may possibly cost her her life.

In the fœtus a series of conditions may occur, the severity of which apparently depends on the amount of antibody and the time at which it becomes effective; these conditions include (i) some types of miscarriage, (ii) *hydrops fœtalis*, (iii) *icterus gravis*, (iv) hæmolytic anæmia.

In the first subgroup mentioned (miscarriage), antibodies affect the fœtus so early and so severely that it dies and is eliminated by abortion in the first half of pregnancy. A somewhat smaller but still very strong effect causes the so-called *hydrops fœtalis*, in which the fœtus is usually stillborn and is characterized by enormous œdema of the soft tissues and the accumulation of fluid in the body cavities. In the third subgroup the baby is born alive and is of normal appearance, but on the first or second day of life develops *icterus gravis*, a deep jaundice, quite distinct from the jaundice that most newborn babies later develop in a mild form. This condition often ends fatally. In the fourth subgroup, the mildest type, the babies are normally developed and do not become jaundiced, but may be very pale. Investigation discloses an anæmia of the hæmolytic type. It has long been felt that the last three apparently totally different syndromes are but manifestations of one underlying condition.

Work on the Rh Factor at the Women's Hospital.

Realizing the importance of detecting as early as possible in their pregnancy mothers who have Rh-negative blood, we have tested the blood of all pregnant patients whose previous history has shown evidence of miscarriage or stillbirth. As shown in Table I, 486 such patients were tested between May and October, 1943, and 101 (21%) were found to have Rh-negative blood. Table I shows the distribution of women with Rh-positive and Rh-negative blood in the different blood groups. It is interesting to compare these figures with those of

TABLE I.

Blood Group.	The Women's Hospital, May to October, 1943. (486 Subjects.)[1]			Landsteiner and Wiener. (448 Subjects.)[1]		
	Rh-Positive.	Rh-Negative.	Total.	Rh-Positive.	Rh-Negative.	Total.
Group AB ..	5	1	6	11	2	13
Group A ..	149	48	197	148	27	175
Group B ..	46	15	61	29	11	40
Group O ..	184	38	222	191	29	220

[1] Part of a symposium on the Rh factor held by the Victorian Society for Pathology and Experimental Medicine on December 13, 1943, at Melbourne.

[1] In the Women's Hospital series, the number of subjects with Rh-negative blood was 101 (21%), in Landsteiner and Wiener's series the number was 69 (15·4%).

Landsteiner and Wiener. The total number of tests and the distribution of the subjects in the different blood groups is of the same order in the two sets of observations. It is noteworthy that among these selected patients in our series there is a higher incidence of women with Rh-negative blood—21% as against 15%.

If our assumption that a certain proportion of miscarriages is due to the activity of the Rh factor was correct, a much greater difference between the figures obtained in our selected series of patients and those of Landsteiner and Wiener might have been expected. However, a great number of the abortions in our series have probably been self-induced, in spite of denials by the patients, and many of the abortions and stillbirths must have been caused by gynæcological disorders. The difference observed is, therefore, of an order which could reasonably be expected, and which strongly supports our assumption.

If the ratio of patients with Rh-negative blood in each group to the total number of patients with Rh-negative blood is expressed as a percentage, it is interesting to note that there is an appreciably higher percentage in group A in our series. This is not due to the occurrence in the series of an abnormal number of persons belonging to group A (Tables IIA and IIB).

TABLE IIA.
Incidence of Subjects with Rh-negative Blood in Each Group Expressed as a Percentage of the Total Number of Subjects with Rh-negative Blood.

Blood Group.	The Women's Hospital, 1943.	Landsteiner and Wiener.
AB	1%	3%
A	48%	39%
B	15%	15·9%
O	38%	42%

TABLE IIB.
Distribution of Blood Groups in Both Series.

Blood Group.	The Women's Hospital, 1943.	Landsteiner and Wiener.
AB	1%	3%
A	41%	39%
B	12%	9%
O	46%	49%

Among these mothers with Rh-negative blood, in forty cases the present pregnancy has terminated. Twenty-four babies have survived; 21 of them are apparently normal, and three after developing *icterus gravis* have recovered. There have been six miscarriages and eight stillbirths, and two babies, although born alive, developed *icterus gravis* and died.

The presence or absence of the Rh factor in the blood of seventeen of the surviving babies has been determined; the blood of six is Rh-negative and that of eleven Rh-positive. Three of the eleven babies with Rh-positive blood developed *icterus gravis*, and the remaining eight may have had a mild hæmolytic anæmia.

Tests for Rh antibodies have been performed before, during and after delivery on many of the mothers with Rh-negative blood, and the results are summarized in Table III. Traces of Rh antibodies were found in some of the blood taken before and at delivery; but the only appreciable amounts of Rh antibodies have been found in blood tested during the puerperium. Among the fourteen patients whose blood contained detectable Rh antibodies during the puerperium, that of seven had a low titre and that of the remaining seven a titre sufficiently high for the serum to be used for Rh testing. The pregnancies of those patients whose blood contained a low titre of antibodies resulted in five living babies, one stillbirth and one erythroblastotic child who died. Among the results of pregnancies in which large amounts of antibodies were detected in the mothers' blood, there were four stillborn erythroblastotic babies, living twins (both of whom developed jaundice and one of whom died), and two babies who developed *icterus gravis*; these two were very ill and were given transfusions of Rh-negative blood, and they both survived.

TABLE III.
Results of 130 Tests for Rh Antibodies.

Period.	Antibodies Present. (Number of Subjects.)	Antibodies Absent. (Number of Subjects.)
Ante partum ..	31 (weak)	63
At delivery ..	6 (weak)	9
Eight days or more *post partum* ..	14	7

It is interesting to speculate where the antibodies are stored in the ante-partum period, since they are so seldom detected. It has been suggested that only a slow formation of antibodies takes place, and that these pass through the placenta into the fœtal circulation and are stored in the fœtal tissues. In severe cases the fœtal tissues are not able to cope with the amount of antibodies formed, and the excess attacks the fœtal red blood cells. On the other hand, in *icterus gravis* it would seem that the antibodies stored in the fœtal tissues are released during the first few days of life, and that as a result of the destruction of the infant's red blood cells jaundice develops. A curious feature is the predominance of blood group A among the patients whose serum contains high Rh isoantibody titres. The seven specimens of blood with high titre were all of group A, as were also three of lower titre in the total number of fourteen. The Commonwealth Serum Laboratories have one specimen of serum of very high titre also from a patient in blood group A, as well as a specimen of group O serum with high Rh antibody titre. Since analysing these figures I have found two examples of group B serum with a moderately high titre; each patient had had an erythroblastotic baby. In a recent publication by Taylor and others, 23 of the 44 mothers whose blood was Rh-negative and whose babies were erythroblastotic were in blood group A. In a report by Hoare, four or five mothers whose blood was Rh-negative and who had had erythroblastotic babies were of blood group A.

The Frequency of Erythroblastotic Children from Mating of Persons with Rh-Positive and Rh-Negative Blood.

Since in as many as 13% of all families the mother's blood is Rh-negative and the father's blood is Rh-positive, the chances of isoimmunization in pregnancy against the Rh factor would lead one to expect a much higher incidence of erythroblastosis. A number of reasons have been advanced to account for its low incidence.

1. Not all the babies of a mother with Rh-negative blood and a father with Rh-positive blood have Rh-positive blood. Since the Rh factor is transmitted as a Mendelian dominant, the fate of the children depends on the father's being homozygous or heterozygous for the factor. If the father is homozygous (Rh Rh), all the children will have Rh-positive blood. All are liable to develop erythroblastosis; but factors to be discussed later may prevent its occurrence on one or even more occasions. If, however, the father is heterozygous (Rh rh), there is an equal chance that every child may have either Rh-positive or Rh-negative blood. When the child's blood is Rh-negative, the pregnancy will progress satisfactorily. If the child's blood is Rh-positive, there is the same possibility, as in the first case, that erythroblastosis will occur—again subject to the action of the factors to be described.

2. Not every mother with Rh-negative blood will produce isoantibodies to the Rh factor. It has been generally thought that the fœtal and the maternal circulations are completely separated, and that no red blood corpuscles can pass from one into the other. It seems, however, that fœtal red blood cells do enter the maternal circulation when tips of chorionic villi break off or become necrotic. The antibody formation in the maternal blood seems to depend largely on the extent of such changes occurring in the placenta. Another factor which has to be taken into account is the possible lack of sensitivity of the mother to the Rh substance.

3. The amount of antibodies formed in any one pregnancy varies considerably. If the fœtal tissues are able to deal with the antibodies formed, the red blood cells of the fœtus may escape destruction and a normal living child may be born. On the other hand, the antibodies formed in one pregnancy may persist in the blood of the mother, and particularly if a second pregnancy follows closely, the existing antibodies *plus* those formed in the later pregnancy may have fatal consequences. It is frequently observed that one or two normal children are born, but later pregnancies are all complicated by one or other forms of erythroblastosis.

4. The mildest form of erythroblastosis, the hæmolytic anæmia of the newborn, may not be diagnosed as such unless attention has been directed to the possibility of its presence. Mild forms of erythroblastotic icterus may be classified as a rather severe ordinary *icterus neonatorum* if no special investigations are made. Javert has estimated that erythroblastosis occurs in one out of 400 babies. This figure is probably too low on account of the reasons just discussed. But even if allowance is made for this, the actual figures are much smaller than one might expect from the frequency of matings between persons with Rh-positive and Rh-negative blood. The factors discussed under the first, second and third paragraphs in this section explain this discrepancy.

The Management of Erythroblastosis.

Nothing can, of course, be done to prevent the action of Rh substance from the baby from producing Rh antibodies in the mother, or the passage of these antibodies through the placenta into the fœtal circulation. Nevertheless, testing for Rh factor and for Rh antibodies is necessary for the following reasons.

Transfusion for the Mother.

If a serious hæmorrhage occurs during the course of pregnancy or at delivery, it may be necessary to give a blood transfusion to the mother. The use of Rh-positive blood may well prove fatal; the Rh antibodies in the patient's serum may cause destruction of the donor's red cells, with the same reactions and consequences as in the usual incompatible blood transfusions. If it is previously established that the mother's blood is Rh-negative, Rh-negative blood free from antibodies can be used and such reactions avoided. In obstetrical cases the hæmorrhage is usually sudden and extensive. The most effective treatment has been proved to be the rapid transfusion of a large volume of blood (four or five pints within an hour). Obviously direct transfusion from a donor will not meet the case. Recent discussions with officers of a transfusion service have had the result that large volumes of Rh-negative blood are available immediately.

The Child.

Transfusion.—If the baby is born alive, but develops jaundice or severe anæmia a few hours or days after birth, transfusion of blood from a suitable donor will in most cases result in survival and recovery within a short time. The blood transfusions which have been used in such cases in the past were given, of course, without regard to the Rh factor. Frequently the blood of the mother was used for the transfusion, as being the most convenient to obtain. This procedure has probably killed many of these unfortunate infants, since the Rh antibodies introduced with the mother's transfused blood, in addition to the Rh antibodies in the child's own circulation, have probably been sufficient to destroy, not only practically all of the infant's own red blood cells present at the time of transfusion, but also any newly released cells. Transfusion of Rh-positive blood from the father or some other donor is not likely to be satisfactory, as the Rh antibodies in the baby's circulation will attack the donor's cells as well as the child's own cells, so that little benefit and possibly considerable harm may result. Hence transfusion of Rh-negative blood, free from antibodies, is the only treatment likely to be effective. In a recent article Wiener and Wexler recommend the use of two transfusions of such blood, each of about 75 cubic centimetres, as being adequate in most instances to provide enough red blood cells to supply the body's requirements until the Rh antibodies have disappeared from the infant's blood and sufficient of its own red cells have been formed. They go so far as to recommend transfusion if there is any evidence of the likelihood that such a condition may exist before clinical evidence of its presence is discovered. They suggest transfusion into the umbilical vein before it is tied and cut. It is of interest that these writers have tested the blood of some babies several days after transfusions of 150 cubic centimetres of blood and have found that the only corpuscles present in the baby's circulation were those of the donor. For example, a mother belonged to group AB and her blood was Rh-negative, the baby belonged to group A, and his blood was Rh-positive, and the donor belonged to group O and his blood was Rh-negative; only group O cells were present when the examination was made. Of course, the original blood group will be restored when the cells of the transfused blood are replaced by the child's own cells, which are released from the blood-forming centres after the elimination of the Rh antibodies. It may be added that when Rh-negative blood is not available, the use of carefully washed maternal blood cells suspended in saline solution or pooled human serum has been recommended.

Nursing.—Witebsky states that the milk of mothers of erythroblastotic babies contains Rh antibodies. He therefore recommends that such babies should be artificially fed, since nursing by the mother is likely to increase the Rh antibodies in the child and lessen its chances of recovery. We have tested milk from three mothers whose babies developed *icterus gravis* and found evidence of traces of Rh antibodies.

Advice Regarding Future Pregnancies.

In families in which the mother's blood is Rh-negative and the father's Rh-positive, and all the pregnancies have been unsatisfactory, with the inclusion of those families in which the first pregnancy has been the only normal one, there is little if any chance that a later pregnancy will result in the birth of a living child.

When the obstetrical history shows that normal pregnancies have occurred among abnormal ones, and particularly if any of the surviving children have been proved to have Rh-negative blood, there is an equal chance that any future pregnancies will be satisfactory. In such cases pregnancies should not occur at close intervals, particularly if the previous one has ended in miscarriage or erythroblastosis, on account of the danger that the additive effect of residual Rh antibodies from the previous pregnancy will be superimposed on that of newly formed antibodies in the later pregnancy.

Conclusion.

Finally, investigations in this hospital during six months have detected 101 mothers with Rh-negative blood among 486 women with a previous obstetrical history of miscarriage or stillbirth. The value of warning medical officers that there is the chance that erythroblastosis will complicate some pregnancies is unquestionable. The investigation should be extended to include, not only women whose past history suggests the occurrence of erythroblastosis, but all women attending ante-natal clinics at maternity hospitals. Thus possible tragedies and serious psychological shocks may be avoided among *primiparæ* and among those whose previous pregnancies have been normal,

but who have been building up an immunity. The question of the production of sufficient suitable typing serum is all-important. We are at present dependent on finding suitable serum of sufficiently high titre and polyvalency to detect the maximum number of mothers with Rh-negative blood. Many difficulties are experienced in obtaining blood from the patients, even when a high-titred serum has been detected. Firstly, we are faced with the fact that the titre of antibodies usually decreases rapidly. This necessitates the taking of blood from the patient within a week or two after her confinement. The patient herself is frequently reluctant to consent to undergoing the unpleasantness associated with the removal of a large volume of blood so soon after confinement. The doctor also frequently feels that the loss of a pint of blood at this stage may not be in the best interests of his patient. Coupled with the uncertainty that sufficient suitable serum will be available, there is the question of variability of titre and "polyvalency" in serum from these patients. The production of a standardized product such as has been prepared for the Wassermann and other tests would gladden the hearts of workers in this field.

THE RH FACTOR: ETHNOLOGICAL ASPECTS.[1]

By R. T. SIMMONS *et alii*,[2]

From the Commonwealth Serum Laboratories, Melbourne.

I SHOULD like to summarize briefly the results obtained in five separate blood investigations on four different and coloured races. The detailed results of these surveys have not yet been published. Different workers have been involved in the investigations, and the persons concerned were Dr. Lucy Bryce, Mr. J. J. Graydon, Dr. Harold Wilson, Miss Patricia Hamilton and myself. The investigations cover the blood groups O, A, B and AB, the subgroups A_1, A_2, A_1B, A_2B, the M, N types, M, N and MN, and the Rh factor. These surveys were made possible by the generous cooperation of field collaborators.

When we first decided to undertake this work we had blood grouping serum available, and the M and N testing fluids had been successfully prepared. At the time of the first survey, as far as we know, no large-scale investigations had been made on blood samples collected some thousands of miles away from the laboratory. It was impossible for us to make long trips away, and quite useless, in that none of us were anthropologists.

It may be of interest to know just how these investigations were arranged.

We tested blood preserving fluids and found that the glucose-citrate solution of Rous and Turner, to which we added "Merthiolate" to give a concentration of 1 in 100,000, would preserve blood cells for many weeks, even months, provided the blood was taken from the finger well swabbed with ether and that the samples were stored in the refrigerator.

For the first investigation on Australian aborigines, carried out some considerable time ago, we had to locate the best districts from which aboriginal blood samples could be collected—places on air routes—and a responsible person to act as a field collaborator, who would collect the specimens for us—a person who knew the natives and their family history, or who had access to settlement records. This took some time. When all arrangements were made we sent by air freight large "Thermos" flasks containing everything required by the person collecting the specimens. Two drops of blood only were required in small stoppered phials. After collection the specimens were packed in ice in the "Thermos" flasks and returned immediately to us by air. In every instance except one the specimens have arrived in perfect condition. In this instance, blood was collected at Groote Eylandt in the Gulf of Carpentaria, and owing to collection difficulties some of the specimens were stored for about eighteen days before being sent on to us; some of these specimens showed pan-agglutination when tested.

Just after this investigation was commenced and samples had been obtained from Menindee, New South Wales, a paper was published by Birdsell and Boyd, who unknown to us had through Harvard University and the University of Adelaide arranged an Australian expedition. This expedition, led by Dr. Birdsell, conducted an anthropological survey and examined the blood groups and M, N types of aborigines in certain parts of Australia.

We decided to proceed with our first investigation and to obtain samples of blood from aborigines in parts of Australia not visited by Birdsell.

Earlier work by Professor J. B. Cleland and his colleagues over a period of years in South Australia and the Northern Territory had shown that the 776 blood samples which they had obtained from pure-blooded Australian aborigines all belonged to group A or group O.

Birdsell examined 805 samples of blood and found that they were mainly of group O or group A. Most of these samples were obtained in Western Australia and the north of Queensland. Amongst their samples they found 2·1% to be of group B, and these natives had all originated in the north of Queensland. Gay examined the blood of 201 natives in the Broome district of Western Australia, and all were of group O and group A. For our first survey we examined 649 blood samples from twelve different places in Australia, and all were of group O or group A.

The M, N typing results were interesting. Birdsell found 67% to be of type N, and this figure was the highest found anywhere in the world. Our figure for samples over a wider area was 51% type N, and in addition we found that subgroup A_2 was absent.

Our second investigation was carried out in the latter part of 1943, after the Rh work had been placed on a satisfactory basis in Melbourne. For this survey we obtained 281 blood samples from aborigines living at Hermannsburg Mission, Oodnadatta, Menindee, Woorabinda and Palm Island. All of the 281 samples were found to be Rh-positive and those of group A not previously tested were found to be of subgroup A_1.

At this point I should like to digress a little and say something of how blood group information is being used.

The blood group and type information which has been collected throughout the world is being used as an aid in the study of racial origins, and fresh information concerning the M, N types, subgroups and the Rh factor should help considerably to build up the picture.

Boyd considers that any "race" which is distinguished on the basis of some constellation of physical characteristics will be essentially artificial, and all sorts of exceptions and gradations between types will be found. Boyd makes the following statement concerning the use of blood group data:

> The fundamental units of racial variability are populations and genes, not the complexes of characters which connote in the popular mind a racial distinction. Much confusion of thought would be avoided if all biologists realized this fact. The geography of genes, not the average pheno-types, must be studied. Only a few attempts to apply this method in practice have been made to date. The most successful one among them is that concerning the blood groups in man.

One of the most graphic methods of representing the results of blood group frequency studies in different regions is by plotting the frequency of each gene at the point corresponding to that locality and then drawing lines of equal frequency, which are referred to as isogenes, through points where the gene-frequency is the same. If enough observations are available so that blank spaces do not appear on the map, and if the data are accurate, a

[1] Part of a symposium on the Rh factor held by the Victorian Society for Pathology and Experimental Medicine on December 13, 1943, at Melbourne.

[2] This contribution to the symposium on the Rh factor was presented by Mr. R. T. Simmons on behalf of the authors listed below at a meeting of the Victorian Society of Pathology and Experimental Medicine on December 13, 1943, at Melbourne. The material presented in the address is from data collected by Mr. J. J. Graydon, Dr. Harold Wilson, Dr. Lucy Bryce, Miss Patricia Hamilton and Mr. R. T. Simmons. In a footnote to Table I reference is made to the papers in the press and those to be published.

regular representation of the geographical trends is obtained. The method was first applied to Australian aboriginal blood-grouping results by Birdsell and Boyd, and their results were later extended by us.

Boyd's published world maps showing the frequency of *q*, the gene for group B, has its highest value in central Asia, has a lower value in central Africa, and then falls off as one travels away from these centres, no matter in what direction, and by the time America and Australia are reached the value of *q* for the aboriginal populations has fallen to zero. Group B is found only in the north of Queensland and is almost certainly a recent importation.

When this graphic method was applied to results obtained in Australia for *p*, the gene for group A, we found that we were able to confirm generally and extend the results of Birdsell. The highest incidence of *p* is in the centre of the continent and the regions of lower incidence of *p* are towards the coast.

Our results for Palm Island, like those of Birdsell, were interesting; group B was found in five of 98 blood samples examined, and in addition we found that subgroup A_2 was absent. The aborigines at this settlement congregate there from all parts of the north of Queensland.

If we assume—and there are grounds for this assumption—that the B factor in aborigines on Palm Island was derived from comparatively recent Melanesian admixture, probably from the New Guinea area where *q*, the gene for group B, is 0·1, then the racial complex represented in Palm Island must be one-quarter to one-third Melanesian. If the frequency of the subgroup A_2 in the Melanesian is comparable with that found in most of the races so far examined, one would expect to find some aborigines at Palm Island belonging to subgroup A_2. As none was found, a forecast of a low frequency of the subgroup A_2 in the blood of New Guinea natives was made by us.

The third investigation dealt with blood samples obtained from Papuans, 200 samples in all being obtained from natives who had originated in 179 villages. Thus an excellent sampling was obtained. The samples were sent to us by Lieutenant-Colonel Mack and Major Ferris.

The blood groups for different parts of New Guinea have been tested by other workers; but no samples had been subgrouped or tested for the M, N or Rh factors. We found that the four blood groups were present, the incidence of group B being 19% and that of group AB 7·0%. Subgroup A_2 was absent. This finding confirmed the prediction made following our results on Palm Island. The type N percentage incidence of type N was found to be 69; it is thus the highest percentage found anywhere in the world and takes from the Australian aborigine the place of honour. In this instance, as in the case of Australian aborigines, all of the 200 samples were Rh-positive.

The next investigation, which is incomplete, deals with pure-blooded Fijians. We obtained through Dr. Godfrey Barnes, of the Medical Department, Suva, the first collection of 100 samples from pure-blooded Fijians, the natives concerned having originated in 93 villages. All blood groups were found; the incidence of Group B was 18%, subgroup A_2 was absent, the incidence of type N was 50% (approximately the same as in the Australian aborigines), and once again all samples were Rh-positive.

The last investigation, and the fifth on the list, was commenced last week. Blood samples from 239 Indonesians—mostly from Java—have been obtained in collaboration with Dr. Ouwehand, of the Netherlands East Indies Forces. This investigation is proceeding.

The four blood groups were found as expected; the incidence of group B was 22·2%, and subgroup A_2 was absent. The M, N results are most interesting. The N and MN percentages reverse those found by Postmus, whose results were regarded as unreliable ($D/\theta = 7·67$). In addition we found only two samples in 239 which lacked the Rh factor, and these may be due to some untraceable white admixture. The Rh-positive percentage is thus 99·2.

Table I shows the results of the various surveys quoted, and surveys of other races not examined by us are included for the purpose of comparison. The figures shown by me for our work are given with the permission of the various authors, and as most of our surveys are incomplete the figures are subject to alteration when more data are collected and the material is finally submitted for publication.

From Table I it will be seen that pure-blooded Australian aborigines over a wide area of Australia lack the B factor, that subgroup A_2 is absent, that the type N percentage is exceptionally high and that all samples of blood are Rh-positive. The results found for the American Indians are similar, except that they possess almost the highest type M percentage in the world. The Papuans possess the B factor, subgroup A_2 is absent, yet reported, their type N percentage is the highest in the world, and the blood of all of them is Rh-positive. The Fijians also possess the B factor, subgroup A_2 is absent, the type N percentage is high, and the blood of all of them is Rh-positive. The Indonesian group, Javanese *et cetera*, also possess the B factor, subgroup A_2 is absent, and our figures for types N and MN reverse those quoted by Postmus. Here again all samples of blood were Rh-positive, with two exceptions out of 239. The Chinese have the B factor, subgroup A_2 is present, but the percentage is very low or absent, and all samples of blood are Rh-positive with one exception in 150. The Negroes examined in the United States of America are particularly interesting, in that they possess an extremely high percentage of subgroup A_2, and their Rh-positive percentage falls between that for whites and that for the other coloured races examined.

For the purpose of comparison, the figures found for Australian whites are quoted. Reference to the authors concerned in the surveys conducted in Melbourne is made in a footnote to Table I.

TABLE I.

Investigators.	Race.	Number Tested.	Group. (Percentage.)				A_2 (Percentage.)	Number Tested.	MN Types. (Percentage.)			Rh Factor Present. (Percentage.)
			O.	A.	B.	AB.			M.	MN.	N.	
Cleland *et alii* ..	Australian aborigines.	776	38·5	61·5	0	0						
Birdsell and Boyd	Australian aborigines.	805	53·2	44·7	2·1	0		730	3·0	29·6	67·4	
Gay	Australian aborigines.	201	61·2	38·8	0	0						
Melbourne 1[1] ..	Australian aborigines.	649	56·1	43·9	0	0	0	649	11·1	37·3	51·6	
Melbourne 2 ..	Australian aborigines.	281	45·6	52·7	1·7	0	0	70 M, N 281 Rh	10·0	40·0	50·0	100
Landsteiner *et alii*	American Indians.	120	73·3	25·8	0·8	0	0	120	56·7	39·2	4·2	99·2
Melbourne 3 ..	Papuans.	200	45·5	28·5	19·0	7·0	0	200	7·0	24·0	69·0	100
Melbourne 4 ..	Fijians.	100	47·0	34·0	18·0	1·0	0	100	5·0	45·0	50·0	100
Melbourne 5 ..	Javanese *et cetera*.	239	50·2	22·2	22·2	5·4	0	239	32·6	44·8	22·6	99·2
Postmus	Javanese *et cetera*.	194	40·0	18·5	35·0	6·5		194	36·0	14·0	50·0	
Levine and Wong	Chinese, N.Y.	150	30·0	34·0	25·3	10·7	3·0	150	23·3	54·7	22·0	99·3
Various authors ..	Negroes, U.S.A.	730	44·2	30·3	21·8	3·7	37·1	97 M, N 113 Rh	29·9	53·6	16·5	92·0
Melbourne and V.R.C.T.S.[2]	Australian whites.[2]		47·7	39·6	9·8	2·9	18·6	1,000 M, N 3,641 Rh	30·6	47·7	21·7	83·4

[1] Melbourne 1: Wilson, Graydon, Simmons, Bryce (to be published); Melbourne 2: Simmons, Graydon, Hamilton (in the press); Melbourne 3: Graydon, Simmons (to be published); Melbourne 4 and 5: incomplete data (to be published).

[2] Whites: Grouping figures quoted with permission of the Victorian Red Cross Transfusion Service; M, N figures (published); Rh figures (published).

TABLE II.

Investigation of Forty-six Mongol and Epileptic Children; Results Expressed in Numbers.

	Number Tested.	Group.						M, N Types.			Rh Factor.	
		O.	A_1.	A_2.	B.	A_1B.	A_2B.	M.	N.	MN.	Rh+	Rh−.
Mongols ..	24	10	8	1	2	2	1	7	5	12	19	5
Epileptic ..	22	14	4	2	1	1	0	6	3	13	19	3
		O.	A.		B.	AB.		M.	N.	MN.	Rh+.	Rh−.
Totals ..	46	24	15		3	4		13	8	25	38	8

There is one other small investigation which I would like to mention for the benefit of the clinicians present. This investigation was carried out at the request of Dr. S. J. Cantor, of the Children's Cottages, Kew. He sent us 46 blood samples from epileptic and mongol children, and we found that the blood groups, subgroups, the MN types and the Rh factor percentages did not differ significantly from those found in the normal population. The results of this small survey are given in Table II and are published with the permission of Dr. Cantor.

An opportunity was presented in the present circumstances for the complete blood-grouping examination of certain races, and it may never occur again. In spite of heavy commitments of work in other directions, these investigations were undertaken; moreover, the information we have gained in these surveys should prove of definite value, as some of the races concerned are actually in war zones.

The anti-Rh human serum found by the team (I refer to the Victorian Red Cross Transfusion Service, the Women's Hospital and the Commonwealth Serum Laboratories) has been used in Melbourne for work of a high order, as you will have gathered from the previous speakers. In addition, we are now helping laboratories in all States with serum and advice. The small stocks of serum available at present are being distributed in small quantities, as a free service, by the Director of the Commonwealth Serum Laboratories to major hospitals—maternity, military and civilian—where they will be used to the greatest advantage of the community as a whole.

Finally, if the various data we obtain are added to the common pool, we may yet obtain a pattern from which the answer to the question of racial origins may be found.

A TIME-SAVING MASTOIDECTOMY DRESSING.

By J. R. HUTCHEON, M.R.C.S., L.R.C.P., D.L.O. (England),
Major, Ear, Nose and Throat Specialist at an Australian General Hospital.

THE healing of infected wounds of bone has always presented a problem in surgery, in view of the slowness of the process and the consequent long stay of the patient both in hospital and in the out-patient department afterwards. This is particularly applicable to the healing of the cavity produced by the Schwartze mastoid operation. Here the usual method of after-treatment is to leave the bony mastoid cavity open after operation, and to allow it slowly to granulate up from the bottom by packing it at intervals of four or five days with iodoform or "Bipp" gauze. By this method, the cavity takes anything from six to ten weeks (sometimes even more) to fill in, and although the patient need not be in hospital all this time, he certainly has to attend the out-patient department regularly until no more dressings need to be done—that is, till the cavity has filled right in.

In civilian life this is bad enough; but in an army hospital, where patients must be retained in hospital until their wounds are completely healed, the time wasted is of great consequence. Hence it is not to be wondered at that efforts have been made to shorten the time of healing of these bony wounds. One method, which certainly somewhat shortens the time, is to suture together the upper portion of the wound at operation, leaving a small lower portion open for drainage and for packing with the usual "Bipp" gauze, or for insertion of rubber drainage tubes.

A much greater saving in time is obtained by the use of the so-called blood clot method. In this method, the operation cavity is smeared with "Bipp" of a creamy consistency and allowed to fill with blood clot; the edges of the wound are then completely sutured together along their whole length. The wound is completely healed in two weeks, which is about the theoretical minimum time for the repair of such a wound. However, this ideal time-saving result is achieved only in a small percentage of cases in which this method is tried, and therefore it is of use only in certain selected cases of Schwartze mastoidectomy—those cases of subacute mastoiditis in which the otorrhœa has been persisting for five or six weeks or more in spite of efficient treatment, and unassociated with undue pain or giddiness or mastoid tenderness, and in which mastoidectomy is performed only to stop the discharge. In nearly all other cases of Schwartze mastoidectomy in which the blood clot method of treatment is used, the wound breaks down at its lower end and commences discharging. Even in the more modern method of filling the wound with sulphanilamide powder before completely suturing it, the wound often breaks down at the lower end. Then the cavity has to be packed regularly with "Bipp" gauze in the usual way, and takes the usual six weeks or more to fill in.

Obviously, if we could apply the blood clot method in all operations of this type, we should have a solution of our problem of saving time. Why, then, does the method fail in the great majority of cases? The answer, of course, is that the considerable amount of discharge from the primary infection of the bone has to drain away, and it can do this only by breaking down the previously sutured wound. The successful results in this blood clot method are usually obtained only in mild and subacute mastoiditis, in which there is a small amount of discharge from the primary bone infection—small enough to be absorbed within the wound. The complete suturing of the wound then prevents any secondary infection, and the wound heals rapidly. In the more severe and more acute cases of mastoiditis, in which the primary bone infection causes considerable discharge, the mere breaking-down of the wound to allow egress of this discharge at once opens up a path for secondary infection of the wound; this causes a prolongation of the discharge, which delays healing for six or eight weeks or more (exactly as the usual procedure of leaving the wound open and packing it with iodoform gauze leaves a path open for secondary infection).

Thus it will be seen that what we require to solve the problem and to allow all these mastoid wounds to heal in a period of two to three weeks is a method of dressing by which all discharges will be absorbed from a wound left partially open (that is, sutured at its upper end only), and yet which will not allow the entrance of secondary infecting organisms to prolong the discharge. At first this would seem extraordinarily difficult; but if we go back

through medical history a little to the time of Joseph Lister, we find that he advocated the very method for which we are searching.

In his book,[1] Lister laid down the hypothesis that the entrance of secondary infection prolonged the healing time of wounds, and he proved this completely by the rapid healing of a large series of infected wounds in which he effectively prevented the ingress of secondary infection (that is, from the outside). Unfortunately, a great deal of his teaching has been forgotten in the last half-century. The general impression nowadays seems to be that he advocated the use of antiseptics in an open wound. Reference to his book, however, will show that nothing was farther from his mind, and that what he did advocate was the use of antiseptics to prevent the secondary infection of a wound; but he was very careful, after a preliminary swabbing-out of the operation cavity with a weak phenol solution (1 in 40), never to allow the antiseptic solution to touch that wound again. As he pointed out, an antiseptic, being somewhat irritant, would cause inflammation on its own account and thus produce extra discharge. He was always looking for an antiseptic which would be completely non-irritant to the deep tissues.

For the last twelve months I have been applying Lister's principles of dressing of an infected wound in my Schwartze mastoidectomy cases. At first gauze impregnated with dry boric acid was used (ordinary boric lint would have done), as advocated by A. C. F. Halford in his book "Lister Redivivus",[2] and this certainly gave excellent results, although occasionally even the boric acid proved a little irritant. However, in the last few years there has appeared on the market a trade preparation of silver manganite known as "Simanite", which is held to be a completely non-irritant, fairly potent antiseptic, and for the last eight or nine months this has been used with complete success. In all cases so far in which this preparation has been used there has not been the faintest sign of irritation of the tissues.

In the cases under my care, the dressing of Schwartze mastoidectomy wounds has been carried out as a routine procedure. As soon as the operation is completed, the cavity is syringed out with sterile saline solution to remove any bone chips, and then swabbed out with a 1 in 40 solution of phenol; the upper half of the wound is then sutured, the lower half being left open. A piece of dry "Simanite" impregnated gauze, about four or five layers thick, is then soaked in "Simanite" colloidal suspension and applied directly to the wound. This piece is then covered by about eight to twelve thicknesses of dry "Simanite" gauze, and the whole is bandaged tightly in the usual way.

In the ward, the routine is as follows: twice a day the ward sister lifts the bandage and inspects the outer layer of gauze; the moment any blood or discharge appears on its outer surface (as will nearly always be the case in the first twenty-four hours), the outer five or six layers are removed, the inner layers being kept carefully in place all the time on the wound, and five or six fresh layers of dry "Simanite" gauze are applied over the top of the inner layers. This procedure is repeated the moment any further discharge appears on the surface of the gauze.

Apart from this simple routine, there are three occasions when a complete dressing is done: on the third, sixth and fourteenth days. On the third day it is done to remove the blood-stained and stiffened lower layers of gauze which by their hardness are becoming uncomfortable to the patient; on the sixth day, it is done for the purpose of removing the few sutures at the upper end of the wound; and on the fourteenth day it is done to allow inspection of the wound. If this has healed and a firm scab has formed on it, the dressings are no longer required. On each occasion on which a complete dressing is indicated, the following procedure is adopted.

All gauze is removed and any discharge adhering to the wound edges or skin is carefully sponged away with cotton wool dipped in a 1 in 40 solution of phenol, care being taken to prevent any of the phenol dropping into the cavity itself. About five layers of "Simanite" gauze are then soaked in "Simanite" colloidal suspension and applied to the wound, and then covered with more dry "Simanite" gauze. Finally the bandage is applied.

At the end of fourteen days, the whole dressing is removed, and in most cases the wound is found to be healed and covered by a dry scab. If this is so, the dressing can be left off altogether and the patient is ready for discharge from hospital to his work. If the wound is still a little open and is discharging, the complete dressing must be reapplied as already described. Usually, if the wound has not completely healed over by this time, small granulations will be found at the edges of the wound, and these should be burnt right down with pure silver nitrate before the dressing is applied. After another four days the gauze is again removed, and if the wound is healed, it is left off. If the wound is not healed, further dressings are applied till it is.

During this post-operative period, the auditory meatus is treated in the usual way—drops of *Spiritus Vini Rectificatus* are instilled twice a day, and by about the fourth to sixth day the discharge from the meatus usually ceases. Thereafter no drops are needed.

Nine patients have now been treated by this method, and the results obtained have amply justified its adoption. These cases are briefly recorded here; one case (number five on the list) is an exception, which appropriately proves the rule.

Reports of Cases.

CASE I.—C.E.A. suffered from subacute mastoiditis, with a history of left otorrhœa of eight weeks' duration. On February 16, 1943, a Schwartze mastoidectomy was performed on the left side, and a large cavity full of mucopus was found just posterior to the antrum; the antrum itself was full of thick, glairy, mucoid discharge. The routine dressing was applied, and in fourteen days the wound was completely healed and the ear was dry. The patient was discharged from hospital fit for duty the next day.

Of course, this was a subacute type of mastoiditis, and the wound might have healed well in the same time by the blood clot method.

CASE II.—M.McC. had fairly acute mastoiditis with a history of otorrhœa of three and a half weeks' duration, and in the last five or six days, increasing pain and tenderness had been felt in the mastoid area, particularly at the tip. Schwartze mastoidectomy was performed on the right side on April 5, 1943, and considerable pus and granulation were found all through the cells of the mastoid tip. Pus was also present in the cells at the root of the zygoma and in the mastoid antrum. In this case the stitches were removed a little later than usual (eighth day), and at this dressing the wound was found healed except for three small areas along its length. The dressings were removed on the fourteenth day, and the wound was found to be completely healed and the ear dry. The patient was ready for duty next day.

This case was one in which healing would have been very unlikely to be complete in so short a time if the usual after-treatment of the blood-clot method had been adopted.

CASE III.—W.T.A. had had a Schwartze mastoidectomy performed in a civilian hospital four months previously, and had suffered recurrent attacks of pain and tenderness at the mastoid tip ever since. On May 6, 1943, the mastoid was reopened; all the cells at the mastoid tip were found still present and full of thick, mucoid material. The routine dressing was applied, and in twelve days the wound was healed and the ear dry. The patient was ready for duty in fourteen days.

CASE IV.—J.M.G. had a four weeks' history of left otorrhœa with a fair amount of mastoid pain and some tinnitus. Schwartze mastoidectomy was performed on the left side on June 3, 1943, and pus and granulation were found both in the antrum and in the cells external and posterior to the antrum. The routine dressing was applied, and the wound was healed and the ear dry in twelve days. The patient went back to duty in fourteen days.

CASE V.—H.J.P. had a history of acute mastoiditis of three days' duration, associated with severe pain and otorrhœa, some giddiness, and extreme tenderness over the left mastoid antrum. On February 4, 1943, Schwartze mastoidectomy was performed on the left side; pus was found all through the mastoid cells right down to the tip, where some particularly

large cells were found. In addition, an extradural abscess was found in the middle cranial fossa. The routine dressing was applied and fourteen days later the ear was dry, but the posterior wound continued to discharge through a fair-sized sinus in spite of the application of the routine dressing for another five weeks. However, suspicions were now aroused that all was not right, and on a close watch being kept on the patient, it was discovered that on occasions he was pushing his finger in under the dressing onto the bare wound "to see how it was progressing". He was warned to discontinue this practice, and the routine dressings were reapplied; the sinus closed and the wound healed in a further six days. The ear had remained dry all the time, and he was discharged from hospital to duty eight weeks after operation.

The point of interest in this case is that the patient was introducing secondary infection into the wound. As soon as his efforts were curtailed and secondary infection was rigidly excluded, the wound healed rapidly. The post-operative duration in this case—from operation to duty in eight weeks—was, of course, no improvement on the normal method of packing the wound; but the history illustrates the importance of excluding secondary infection from these wounds.

CASE VI.—N.C.S. had a history of fairly acute mastoiditis. For three weeks he had had a profuse discharge from his left ear, with occasional aching pain behind the ear, and in the last few days a fair amount of rotatory giddiness. On June 29, 1943, Schwartze mastoidectomy was performed on the left side, and an extradural abscess of the middle cranial fossa was found, as well as a large cavity just behind the antrum containing thick, glairy mucopus. The usual dressing was applied, and at the fourteenth day the ear was dry and the wound was almost healed, though it was still a little open and discharging at one spot where there was a mass of granulation. This was burnt down with silver nitrate and the dressing was reapplied. The patient was discharged to his unit with his wound healed and his ear dry in twenty-one days from operation.

CASE VII.—B.F.T. had an unusual history. He was admitted to hospital on June 16, 1943, with a history of pain and deafness in the left ear, but no discharge, of two weeks' duration. On examination, he was seen to have a papilloma of the posterior meatal wall with much dammed-up wax behind it. He was given a general anæsthetic and the papilloma and wax were removed. The lower layers of the wax presented a cholesteatomatous appearance. From then on the ear discharged pus continuously, and it seems reasonable to assume that this was due to chronic *otitis media* and that infection had been present in it for months. The ear continued to discharge profusely in spite of the use of spirit drops and a course of sulphathiazole tablets, so on July 29, 1943, Schwartze mastoidectomy was performed on the left side, and mucopus and much granulation were found in the antrum, the mastoid process itself being dense and acellular.

Really, if it was assumed that chronic *otitis media* was present in this case, a radical or modified radical mastoid operation was indicated; but it has been found in practice that in a number of these cases of moderately chronic otorrhœa, so long as the discharge has not been continuing for too long, the condition will settle down slowly after a simple mastoidectomy. The operation can easily be converted into a radical operation later if the discharge does not settle down in a reasonable time.

In this case the routine dressing was carried out, and in fourteen days the wound was healed and dressings were left off. However, the ear was still discharging to a slight extent, though much less than previously. The treatment was changed from the use of drops of *Spiritus Vini Rectificatus* to the daily insufflation of iodoform and boric powder into the ear, and in another six days the discharge had ceased. Treatment was then stopped and the ear watched for another five days; but it remained dry. The patient was discharged from hospital to duty twenty-five days from operation.

CASE VIII.—D.E.M, was another patient with right otorrhœa that had reached the chronic stage. The ear had been discharging for nine weeks when he was admitted to hospital. He was treated for another two weeks with drops and with sulphathiazole tablets, but no reduction in the amount of the discharge took place, so on October 14, 1943, Schwartze mastoidectomy was performed on the right side. The dense, acellular mastoid was found that is usually characteristic of cases of continued otorrhœa without any symptoms of pain or giddiness. There were a few cells immediately posterior to the antrum, and these and the antrum contained pus and much granulation. The routine dressing was applied, and in fourteen days the wound was healed. The discharge from the ear was much diminished, but it still continued, slowly decreasing under treatment with drops of *Spiritus Vini Rectificatus*, until it finally ceased in a further five weeks.

CASE IX.—M.F.R. had a history of acute mastoiditis. For nine days he had suffered from profuse otorrhœa with pulsation and much pain and mastoid tip tenderness. On October 14, 1943, Schwartze mastoidectomy was performed on the right side, and all the cells of the mastoid tip were found to contain pus, as well as a large group of cells in the posterior superior angle. The usual dressing was applied, and on the fourteenth day the wound was completely healed and the ear dry. The patient was discharged from hospital the next day.

This series of nine cases includes a wide variety of different histories in which Schwartze mastoidectomy was indicated. There are examples of extremely acute mastoiditis, moderately acute mastoiditis, subacute mastoiditis and even two of mildly chronic otorrhœa. Two cases (Cases V and VI) form an interesting comparison; both patients had extradural abscesses, but one, by introducing secondary infection, prolonged his stay in hospital by many weeks. The other, in whose case secondary infection was rigidly excluded, was fit for duty in twenty-one days from operation. This comparison adds further proof to the contention that it is the exclusion of secondary infection from these wounds that is the *sine qua non* in their rapid healing.

Summing up, we find that the great majority of these patients were discharged to duty in two or three weeks; one patient with a chronic lesion took three and a half weeks, though his wound was healed in two weeks; one patient with a chronic lesion took seven weeks, though his wound also was healed in two weeks; and one patient took eight weeks, though this was not the fault of the dressing. These results seem to justify the use of this technique of dressing mastoidectomy wounds as a means of saving valuable time in the patient's convalescence.

It would seem that orthopædic surgeons should find a useful application of this technique in the treatment of compound fracture and osteomyelitis, which usually require a long period of after-treatment. In fact, this is exactly the type of case in which Lister obtained such excellent results in rapid healing of infected wounds of bone.

Summary.

1. A new method of dressing for mastoidectomy (Schwartze) wounds is described.

2. Nine cases are recorded in which this dressing has been used.

3. A great saving in the time taken in after-treatment is shown, resulting in the speeding of the individual back either to his civilian occupation or to his army post.

Acknowledgements.

I should like to express my thanks to the Director-General of Medical Services for permission to publish this report, and also my grateful thanks to Dr. A. C. F. Halford, of Brisbane, who stimulated me to read Lister's works, and to apply his principles to the mastoid problem.

References.

(1) "The Collected Papers of Joseph, Baron Lister", Volume II, Part 3, pages 38, 51, 323 and 339.
(2) A. C. F. Halford: "Lister Redivivus."

Reports of Cases.

CANCER OF THE FEMALE URETHRA.

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CARCINOMA of the female urethra is a rare disease. Some cases are here presented in which the patients are living normal lives some years after treatment; these cases may be fairly considered as cures. Fourteen further cases are

presented; ten of the patients were married, two were widows and two were single. The average age was fifty-two years. Of the fourteen patients, six are well at present, and one, after living twelve years, cannot be traced.

Hæmorrhage was the most consistent symptom, in many cases the only symptom. Difficulty with micturition was a late symptom; it occurred only in advanced cases. Pain was a symptom that developed late.

The difficulty of diagnosis may be appreciated from the frequency with which a carcinoma is diagnosed as a caruncle and submitted to diathermy treatment on more than one occasion before the patient is handed over for proper treatment. Caruncle is a name used to cover several conditions: (i) the rare true caruncle, almost as rare as cancer, associated with exquisite tenderness; (ii) inflammatory conditions, often associated with a greater or less amount of prolapse; (iii) prolapse of the urethral mucosa. Many of the cancer patients had associated severe inflammation and sloughing. Though the tendency to break down and to bleed easily, and the presence of an edge obviously infiltrated with growth make a diagnosis easy in most cases, a biopsy specimen should always be taken.

Thirteen of the lesions were epitheliomata; one was an adenocarcinoma.

The female urethra is four centimetres in length. Except during the passage of fluid, the anterior and posterior walls are in contact. It has a kink or bend about its middle with an obtuse angle open forwards. It has three coats, muscular, mucous and erectile, and the great vascularity of the part probably accounts for its resistance to heavy doses of irradiation. With slight variation, the same dosage and technique have been used in all cases.

A self-retaining catheter was inserted into the bladder; it contained in its distal end a lead cylinder six millimetres in diameter, bored through its centre with a capillary hole. The total diameter of the catheter was seven millimetres. Five or six radon needles were then inserted parallel to the now straight urethra; the needles were approximately one centimetre apart and two millimetres from the urethral canal. The needles were 2·5 centimetres long, contained 5·0 millicuries of radon and were shielded by 0·8 millimetre of gold. The labia were gently drawn together with a silk suture below the catheter to help keep the needles in position for eight days.

Six 2·5 centimetre gold radon needles, screened by 0·8 millimetre of platinum of a strength of 2·0 millicuries per centimetre, were inserted and left for eight days; they were distributed uniformly around a cylinder of a diameter of 11·5 millimetres. The central core was an inner lead of cylinder five millimetres in diameter surrounded by rubber tube, the external diameter being 7·5 millimetres. The treated tissue extended between concentric cylinders of diameters 7·5 and 17·5 millimetres and length 2·5 centimetres.

The average minimum dose around the outside of the treated tissue was 19,000 *r*, while the maximum dose was approximately 40,000 *r* one millimetre away from the surface of the needle. It has been found that a dose of 6,000 *r* is extended through a cylinder of a diameter of about 36 millimetres—that is, through a thickness of tissue of 14 millimetres.

The dose distribution throughout the treated volume is fairly satisfactory; but it could be improved by the use of eight needles containing the same total amount of radon as is used at present in the six needles.

It was found that the lead cylinder made a small difference in the dose received at the various points; but the lead cylinder and the catheter keep the urethra straight and parallel to the needles and also the volume of tissue irradiated is increased.

Some pain on and frequency of micturition follow treatment. These abnormalities usually settle down in six to eight weeks. The patient is given abundant fluids and citrate of potash during this period. Sloughing was not serious except in one case, Case II, in which two lots of irradiation were given. Radon in "Vaseline" was used once a week in this case (eight hours) and was thought to have helped in healing the condition.

Stricture has not been a serious complication except in the above-mentioned case (Case II), and it might perhaps have been expected after the sloughing which took place. Dilatation of the urethra with the aid of a local anæsthetic agent is easy; but Kelly's dilator should be used, not a Hegar's dilator, which does unnecessary damage and seems more inclined to cause incontinence of urine.

If the growth tends to metastasize towards the inguinal glands, block dissection is indicated in suitable cases. In my pre-radium days (before 1929) I have often seen operation wounds of block dissections of the inguinal glands for malignant disease of the vulva sloughing from end to end, with the femoral arteries pulsating in the depth of the wounds. In the case in which a block dissection was performed, it was not carried out until the primary growth had healed. All wounds healed by first intention. Swelling of one or both legs has followed block dissections in the past, and I have always avoided removal of the vertical glands around the saphenous opening. No trouble with œdema of the legs occurred in this series.

Case I.

A single woman, aged fifty-four years, was admitted to hospital on June 12, 1939, with an advanced lesion and a vesico-vaginal fistula. Vaginal examination revealed epithelioma of the urethra and a fistula in the anterior vaginal wall leading into the bladder behind the urethra. Pathological examination revealed epithelioma. The patient was transferred to the Austin Hospital.

Case II.

A married woman, aged fifty-two years, was admitted to hospital on September 1, 1939. Cystoscopic and urethroscopic examination revealed carcinoma of the urethra with secondary involvement of the base of the bladder. Pathological examination of the growth showed it to be an epithelioma. The patient was transferred to the Austin Hospital.

Case III.

A married woman, aged seventy-five years, was admitted to hospital on March 31, 1936. Her past history was not obtained on account of her mental condition. An extensive growth was present around the urethra, which was found to be an epithelioma. On account of the patient's condition, she was transferred to the Royal Park Receiving Home, with the diagnosis of carcinoma of the urethra and senile dementia.

Case IV.

A widow, aged sixty-three years, gave a history of a "lump" on the urethra of eight years' duration. The "lump" had been removed four times with diathermy and once surgically. On November 20, 1939, the standard treatment with radon was begun (31·5 millicuries over a period of 166 hours). On November 25 the patient complained of hæmaturia and pain. A urethroscopic examination was performed. Examination of portion of the tissue removed revealed an epithelioma of transitional type. The radon treatment was repeated. On February 15, 1941, a painful, sloughing area around the urethra was treated with infra-red rays. On March 6 radon in "Vaseline" was applied to the affected areas; one-thirtieth of a millicurie of radon per cubic centimetre was used, and about two ounces of "Vaseline" were required. This was applied to the area and covered with oiled silk, being left for eight hours at each application. On May 1 it was considered possible that some secondary spread to the vulva might have occurred; examination of microscopic sections gave inconclusive results. On May 29 radon in "Vaseline" was again applied to the affected areas; the treatment was given three times at intervals of one week. On July 30 a clean granulating area was present in the region of the urethra.

On November 18 the patient was admitted to hospital with acute retention of urine. The urethra was dilated with a Kelly's dilator under local anæsthesia. On December 9 the patient was well, and had no difficulty with micturition. On May 26, 1942, she was experiencing some difficulty with micturition, and the urethra was dilated. On July 7 she was well. On July 28 the stricture was dilated. On March 2, 1943, the patient was well.

Case V.

A married woman, aged forty-four years, stated that four years previously a caruncle had been excised. She had had diathermy treatment three years and two years before, and hæmorrhage had been occurring at intervals since the last operation.

On her admission to hospital, a growth was found to surround the urethra; it was not tender, but it bled freely. Pathological examination showed it to be an epithelioma. On October 8, 1936, six needles containing 5·0 millicuries of radon were embedded and left for 166 hours. On November 17 she had some pain and a moderately severe local reaction. On December 9 a block dissection of inguinal glands was performed. On February 2, 1937, some sloughing was present around the urethra, but the inguinal wound was well healed. On April 11 a small slough was still present. On September

29, 1937, the patient was well. On December 13, 1938, she was well; only reddening was present around the urethral opening, and it was mostly angiomatous. On March 16, 1943, the patient was well. Some telangiectasis was still present, but she had no discomfort.

Case VI.

A married woman, aged thirty-eight years, was admitted to hospital with a growth surrounding the urethra. She complained of frequency of micturition associated with pain of two years' duration. Pathological examination of the growth showed it to be an epithelioma.

On May 5, 1936, six needles, each containing 5·0 millicuries of radon, were inserted and left for 170 hours. On July 20, 1936, a block dissection of inguinal glands was performed. On August 29 the wound was well healed, and the patient had perfect control of micturition, although she suffered from slight scalding at times. On March 9, 1937, the patient was well; but she had a profuse vaginal discharge from an area of erosion in the *cervix uteri*. On March 18 the eroded area was painted with pure formalin. On October 5 the patient had some hæmaturia, and on December 14 she was well and symptomless. On August 9, 1938, a slight stricture was present; this was dilated. On September 6 she reported that she had had slight hæmaturia since the dilatation, but that this had stopped. On October 4 healing was complete and the patient had perfect control of micturition. On February 3, 1942, she was well.

Case VII.

A married woman, aged thirty-eight years, was admitted to hospital on account of a granular area surrounding the urethra, which bled readily, but was not painful. Pathological examination of a section of tissue revealed epithelioma. On January 31, 1935, radiation treatment was given; five needles were implanted, each containing 6·0 millicuries of radon. On March 26 a block dissection of the inguinal glands was undertaken. On September 12 some urethral hæmorrhage was present; cystoscopic examination revealed a nodule in the trigone, and further treatment was considered. On December 12 the local condition was good, and the nodule in the trigone had disappeared. Some swelling was still present in the inguinal region. On January 14, 1936, the inguinal swelling had disappeared; the patient was well and had no disability or discomfort. The urethra was surrounded by a telangiectatic area, which was not tender. On August 25, 1942, the patient was well.

Case VIII.

A married woman, aged forty-three years, was admitted to hospital on April 5, 1935, with a history of hæmorrhage and painful micturition of about three months' duration. Examination revealed an ulcerating growth surrounding the urethra and extending upwards for about 1·5 centimetres. Seven needles, each containing 5·0 millicuries of radon, were used; but owing to the size of the growth the spacing was wider than usual. On May 21 the patient had no pain, but swelling was present in the irradiated area. On July 9 a block dissection of the inguinal glands was undertaken. On September 3 there was an indolent ulcer in the irradiated area. The Wassermann test was carried out and was repeated after the provocative administration of "Novarsenobillon"; on each occasion the result was negative. On November 26 the ulcer was healing; examination of a section from the edge revealed inflammatory tissue.

On February 17, 1936, the patient complained of pain during micturition. A cystoscopic examination revealed only reddening of the trigonal area of the bladder. This cleared up, and the patient was well and had no pain or difficulty with micturition; she had perfect control. On December 1, 1941, the patient was well, and had perfect control of micturition.

Case IX.

A married woman, aged forty-nine years, was transferred to the radium clinic after diathermy treatment to a caruncle. Pathological examination of a section of the lesion revealed epithelioma. On October 12, 1929, the caruncle was cauterized. On July 3, 1931, portion of the urethra was excised, and examination of a section disclosed epithelioma. On July 16 six needles were inserted, each containing 5·0 millicuries of radon, and left in position for eight days. On August 25 a severe reaction in the inguinal glands was noted; the pathologist could find no evidence of malignant disease. On October 11, 1932, an area of telangiectasis was present around the urethra. The patient was well and had no complaint. On March 11, 1933, she was still well.

On February 8, 1937, she was readmitted to hospital with what were believed to be secondary deposits at the root of the right lung and fluid in the right pleural cavity. On March 1 she was transferred to the Austin Hospital. On May 14, 1943, it was ascertained that the patient had remained well for some time after her discharge from the Austin Hospital, but had been lost sight of. The Government Statist could find no record of her death.

Case X.

A widow, aged fifty-five years, was admitted to hospital on August 25, 1940, complaining of dysuria and frequency of micturition of two months' duration and of hæmorrhage *per vaginam* of three months' duration. A growth of irregular shape was found on one side of the urethra. Pathological examination showed it to be a squamous-celled carcinoma undergoing degeneration. On August 29 treatment with radon was begun; three needles containing 4·0 millicuries (1·5 millicuries *per centum*—a total of 18 millicuries) and three needles containing 2·0 millicuries (1·5 millicuries *per centum*—a total of 9·0 millicuries) were implanted. The total amount of emanation was thus 27 millicuries, and the needles were left in position for 166 hours. On October 28 a block dissection of the inguinal glands was performed. Pathological examination of the glands disclosed a secondary deposit of squamous-celled carcinoma. On January 25, 1941, the patient was discharged from hospital, her condition being unimproved, and on April 10 she died in another hospital.

Case XI.

A married woman, aged forty-six years, was admitted to hospital on February 28, 1934, and was found to have a large growth extending to the base of the bladder. On March 9 she was transferred to the Austin Hospital.

Case XII.

A married woman, aged forty-nine years, was admitted to hospital on September 9, 1940, with the diagnosis of caruncle; she had a history of pain and of hæmorrhage. On September 12 redundant mucosa was removed with the diathermy knife. Pathological examination of tissue revealed adenocarcinoma of the urethra. Eight radon needles, each containing 2·0 millicuries (2·0 millicuries *per centum*—32 millicuries), were inserted and left in position for 166 hours. On November 27 the patient looked well, and on February 17, 1941, an ulcer and a small slough were present at the side of the urethra. On June 17 healing was proceeding, and on August 5 healing was complete. On December 18, 1942, the patient was well.

Case XIII.

A married woman, aged fifty-four years, was admitted to hospital on February 26, 1934, with the diagnosis of urethral caruncle. On February 24 the caruncle was treated with diathermy, and pathological examination showed it to be an epithelioma. Eight radon needles containing 2·0 millicuries (2·0 millicuries *per centum*—32 millicuries) were inserted and left in position for 166 hours. On January 9, 1935, a block dissection of the inguinal glands was performed. Pathological examination of sections of the glands revealed dense fibrosis with foci of lymphatic tissue; no evidence of malignant disease was found. On March 19 the patient was well. On February 10, 1937, she died of carcinomatosis.

Case XIV.

A single woman, aged fifty-four years, was admitted to hospital with a large vesico-vaginal fistula; there was a large hole into the bladder, and no trace of a urethra was found. The history was vague. Examination of sections revealed epithelioma. On July 30, 1939, the patient was transferred to the Austin Hospital.

Summary.

Fourteen cases of urethral cancer are presented. Of interest are the large dose of irradiation given and the retention of urinary control in the cases in which cure was effected.

Acknowledgement.

I desire to acknowledge with thanks the help given by the Commonwealth Radium Laboratory.

Reviews.

MEDICINE AND SOCIOLOGY.

"MEDICINE AND THE SOCIAL ORDER", by Dr. E. P. Dark, is a remarkable book; its second impression has appeared.[1] From a basis of three articles, "Medicine and the Social Order", "Property and Health", "Some Medical Aspects of Crime", all of which have appeared in THE MEDICAL JOURNAL OF AUSTRALIA, the author has wandered widely afield into the causes of war, the problems of peace, education, and medicine in Russia. He deals with his subjects in a broad-minded way, gathers his facts from the far corners of the earth and in Australia, and then delivers his verdict plainly, courageously and clearly. One of the chief merits of the book is that it compels attention even from those who may differ from its "leftish" viewpoint. Dr. Dark pillories the old social order, but in doing so asks those of the "right" to state their case. The map is before our eyes—we must travel to the right or the left. The reader will not be long in doubt before he realizes why the author passes to the left.

Dr. Dark believes that "health is bound up with the political and economic structure of society, so that the medical profession as a whole is inevitably interested in politics and economics, and should speak with authority on all political and sociological questions which have any bearing upon health". In support of this contention he draws attention to the investigations on the relationship between diet, economic status and health. The data are terrible in their implications. That tuberculosis should be so much more common in unskilled labourers than in the more highly endowed, that privileged, well-fed children should be 3·8 inches taller at seventeen than employed children, are some of the pointers to an inescapable conclusion. Medicine and the economic system are closely linked. We must not sit idly to contemplate without action the cycles of depression, the burning of foodstuffs, the social decay which followed in the wake of the last war. Dr. Dark states: "I am convinced that it is time the medical profession became the leaders of the people in all matters that concern the public health."

In the discussion on property, there is mention of the Stockton-on-Tees experiment. Although better housing facilities were given, the result was an increased morbidity. The extra money spent on rent reduced the food allowance, resulting in a 50% increase in the death rate.

Concerning the slum evil, the reader will learn that Australia is not blameless. Already our slums are creating a serious problem in health which can be solved only by a drastic revision of our social outlook. Our author believes that:

> The Government must be told in a way it cannot ignore, that its most fundamental need is for fit men, and that it cannot have fit men until it can assure sufficient food, decent shelter, and work for everyone. This spreading of essential knowledge can be effectively done only by the medical profession through its official associations.

The chapter on "Some Medical Aspects of Crime" is outstanding. Attention is drawn to the dreary monotony of prison life and prison fare, the aim of which is punitive rather than remedial. Dr. Dark points out that in Russia there have been outstanding improvements in the penal code. Its aim is not to deter others, but to re-create citizens and conduct research in criminality.

At first sight the relationship between medicine and the causes of war is not apparent. If, however, it can be shown that economic factors are largely the basis for all war, we are faced with our need to meddle in sociology. Dr. Dark's evidence is intriguing and damning. He considers the Peloponnesian War, the Punic Wars, the Crusades and contemporary history.

It is essential to define the nature of "the State". He uses Laski's thesis that it is merely the coercive power wielded by the dominant group in a country and used primarily to prevent any change that might imperil its power or its profits. In the author's opinion, when capitalism is dominant, the results are predetermined.

> Capitalism, once fully established, has proved to mean over-production and under-consumption; it can produce abundantly, but it cannot get its products distributed. When the home market was glutted the position was temporarily eased by export of goods, capital goods and capital. To get and control the markets for these, the more backward parts of the world were divided up, and monopoly took the place of competition. When this stage is finished and all the world is parcelled out, the inevitable drive to war is clearly manifest, for capitalism must still expand, and it can only do so at the expense of a rival. At the same time the great monopolies must have absolute control of the State to regulate industry to their own advantage, and to reduce the workers once more to serfdom. Here is the inevitable evolutionary chain—competitive capitalism, monopoly, imperialism, Fascism, war.

In discussing peace, Dr. Dark has a vigorous tilt at the federal unionists, but he admits that they have got hold of half the right idea; the other half is socialism—a federal union of socialist States.

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And in conclusion Dr. Dark does not mince his words. We have, he holds, the choice of two alternatives. On the one hand there is capitalism with its profit motive and its psychological accompaniment of unrestrained greed leading eventually to monopoly and fascism. The other alternative is socialism, of which a working model may be viewed in Russia. As practitioners of medicine we have a definite responsibility to make a decision, since much of the evidence lies in the domain of our specialty.

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"Medicine and the Social Order" is a small book which can be conveniently slipped into a bag or large coat pocket. As an interesting week-ender for diversion and stimulus it is strongly recommended. Where there is disagreement, the reader has the pleasure of suggesting workable alternatives.

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The Medical Journal of Australia

SATURDAY, MAY 27, 1944.

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CARRIERS AND ENTERIC FEVER.

WHEN the term "carrier" is used in reference to a particular infectious disease it indicates a "person who has within himself the germs or other infective virus of that infectious disease, but has no other sign or symptom thereof". In enteric fever the ease with which typhoid bacilli in the alimentary canal of a carrier may infect a healthy person needs no emphasis; and the treatment of a patient recovering from the disease will not be complete until it has been determined that he or she is not a carrier. In the account given a week or two ago in these pages of the outbreak of enteric fever that occurred at Moorabbin, Victoria, in March, 1943, the subject of carriers was mentioned. It should, however, be pursued further for several reasons. One reason is contained in the following statement by Dr. F. V. G. Scholes: "If we assume that there still exist one or more undetected persistent carriers, as well as those known and under surveillance, there must be some uneasiness about the possibility of further large or small outbreaks in Melbourne during the next year or two." Again, apart from the general importance of the subject, there is considerable interest in the way in which the carrier question was handled in the Moorabbin epidemic.

In the special report on chronic enteric carriers and their treatment, prepared for the Medical Research Council of Great Britain in 1933 by C. H. Browning and others (see THE MEDICAL JOURNAL OF AUSTRALIA, June 10, 1933) a clear exposition is given. In the first place the available evidence points to the fact that the typhoid bacillus has a very limited capacity for survival in nature apart from the human host, and chronic carriers are therefore one of the chief sources from which fresh epidemics arise. The second consideration of importance is that the spontaneous cure of a chronic carrier practically never occurs. Browning described temporary and permanent or chronic carriers. In convalescence from enteric fever many persons excrete the specific organisms for short periods; these are the temporary carriers. It is of great importance, however, to note that excretion of specific organisms in the fæces may set in some time, even several months, after convalescence. This lateness of onset occurs oftener with enteric bacilluria. Most of those who continue to excrete enteric bacilli for six months after the acute attack will continue to do so. Those who continue to excrete the bacilli after a year will not become cured spontaneously and are to be regarded as permanent or chronic carriers. From another point of view carriers may excrete the organisms from the intestinal tract or in the urine; rarely organisms may be excreted from other sites such as the sinuses. Those who excrete bacilli from the intestinal tract may be biliary carriers or true intestinal carriers or both. That it is important to recognize the true intestinal carrier is sometimes obvious during treatment; it is not unlikely that some of the failures to obtain cures by cholecystectomy may be due to the fact that the patients concerned are true intestinal and not biliary carriers. Browning discusses at some length the problem set by biliary carriers and makes reference to the pathology. The most marked histological changes observed in all gall-bladders consisted of localized and diffuse collections of cells in the mucosa, and these are mainly plasma cells and lymphocytes with scanty eosinophile cells. He refers to cases in which clumps of bacilli are seen on the mucosa of the gall-bladder, but none are found in its substance. He therefore finds considerable support for the opinion of Garbat that in such cases the carrier state is maintained by the gall-bladder acting as "a test-tube containing the bile medium in which the typhoid bacteria propagate without affecting the gall-bladder itself". It is of interest to note that the association of gall-stones with the enteric carrier state is well established. Browning points out that it is seldom possible to decide in any particular instance whether the inflammatory change in the wall of the gall-bladder with the formation of calculi has been initiated by the enteric infection or whether it was on an abnormal mucosa, with or without the presence of gall-stones, that the typhoid bacillus could establish and maintain itself. Even when *Bacillus typhosus* or *Eberthella typhi*, to give the organism its modern name, is found in the centre of calculi in the gall-bladder, this is no indication that the bacterial infection preceded the gall-stone formation, for it is known that the organisms may readily penetrate cracks in stones. In regard to urinary carriers, Browning points out that when the bacilli persist in the urine it is the rule to find in the urinary tract some pathological condition which is accompanied by pyuria. The nidus of infection is situated most commonly in the kidney or the kidney pelvis. In these circumstances surgical treatment is indicated.

Since the carrier state causes the spread of the disease, and since the carrier state is dependent on the presence of lesions such as those mentioned, it would appear quite simple to arrange matters so that no carrier should pass out of control until he had ceased to be dangerous to other people, in other words, until he was no longer a carrier. But this ideal procedure cannot always be carried out. It could not be adopted at Moorabbin and the health authorities displayed both wisdom and common sense in their handling of the situation. Dr. Merrillees states his conviction that, although the carrier state is potentially dangerous, it is not actually dangerous unless (*a*) the carrier has habits which make the infection of other persons likely, and (*b*) unless an opportunity arises for the handing on of the infection. At Moorabbin carriers could not be isolated indefinitely because the general hospitals and the infectious diseases hospital needed beds for other patients. It was decided that the "best" of the

carriers could be discharged to their homes, temporarily at least, to see if their abnormal condition would clear up. Those allowed to go home were those who (*a*) were intelligent and cooperative, (*b*) did not handle food supplies, (*c*) had a private home to go to where conditions were suitable, (*d*) were habitually clean. In regard to the homes, four requirements had to be met: the house had to be sanitary, not crowded and not associated with any public food supply; the house had to be a single private house, not divided into flats and not a boarding house; the drainage and sanitary services had to be effective; all the inmates had to submit to supervision and cooperate with the patient and the health authority. Obviously each carrier had to be considered as a special problem. Very few adults would be detained if they had suitable homes to go to; but infancy, senility and subnormal mentality needed special consideration. Where one man, clean, intelligent and anxious to learn, was released without question, another, somewhat submental, willing to please, but unable to grasp the meaning of the ritual of cleanliness, was persuaded to undergo cholecystectomy with satisfactory results. Carriers released under the conditions mentioned were regarded as released on probation; they are being kept under surveillance and frequent examinations of urine and fæces are made. "Should these persistently show positive the position will be reviewed in say three to six months and other treatment advised or not according to the opinion of consultants."

Expertly though this outbreak was handled, there still remains what Dr. Merrillees calls "grave danger" from missed cases. He points out that though the outbreak has shown the great need for examination of convalescents in regard to their carrier state, it has also shown that the disease can exist in all degrees of severity from an overwhelming toxic disaster down to a trivial, barely noticed indisposition. "No matter how careful we may be in respect to detecting and educating chronic carriers among the convalescents, there is an absolute certainty that some carriers will escape discovery because they never have been patients." Only a superficial view has been taken here of some of the aspects of an enormous subject; the main thing is that practitioners should never allow the idea of a disease greatly lessened in incidence to lead to any complacency about its sporadic appearance. Perhaps one of the most significant statements in Dr. Merrillees's excellent report is contained in the following words: "This outbreak shows we have signally failed so far; we owe this outbreak to a previously undetected carrier working on a dairy farm and with not even the rudiments of hygiene." In the ideally hygienic state of the future no one will be allowed to handle food for distribution until he or she has been certified as free from communicable disease and addicted to clean habits.

Current Comment.

PERITONEOSCOPY.

THOUGH the method of examination known as peritoneoscopy has been known for many years and reliable surgeons have claimed that it has a sphere of usefulness, it has never been widely used. Reports have appeared in the literature from time to time, and in the last year or two these have been more numerous. It was as long ago as 1901 that Kelling first described the method of examining the peritoneal cavity by means of a cystoscope introduced through a small incision in the abdominal wall. Writing in the *British Medical Journal* of August 8, 1925, A. Rendle Short described briefly the advantages of the method by which air is introduced into the peritoneal cavity through a tiny incision made under local anæsthesia and a cystoscope is introduced for the inspection of the viscera. He stated that the method (he used the term cœlioscopy) did not and could not replace exploratory laparotomy when the cause of a pathological condition might have to be sought which did not lie on the surface of the abdominal viscera. These he thought would include the majority of lesions. The method, he declared, was principally valuable for what was definitely seen and not for what was apparently absent. Short remarked that it was like every other surgical procedure in that a little trouble had to be taken to learn how to obtain a good view, but it was no more difficult than cystoscopy. R. M. Walker and P. L. Playfair, writing in *The Lancet* of February 7, 1942, make much the same kind of remark. At first a cystoscope was used for this work, but a special peritoneoscope has been evolved and is generally used. It is inserted after a pneumoperitoneum has been produced with air introduced through a special trocar. In addition J. E. Hamilton has described in *Surgery, Gynecology and Obstetrics* of February, 1942, a new "manipulator" which may be used in conjunction with the peritoneoscope. It consists of a blunt-tipped rod, the manipulator proper, and an insulated endothermy electrode, both of which fit into a cannula such as is used with a trocar for paracentesis. The trocar and cannula are introduced through a separate stab incision under vision with the peritoneoscope; the trocar is then removed and the manipulator or the electrode may be inserted. The manipulator is used to push on one side structures that obstruct the view through the peritoneoscope.

E. B. Benedict, writing from the Massachusetts General Hospital, has recently reported experiences with peritoneoscopy in liver disease.[1] In a series of 435 peritoneoscopy operations the question of liver disease arose in 331 or 70%. Among the 435 cases there were three in which death was probably caused by the procedure. One death resulted from heart failure and was that of a patient who suffered from multiple lung abscesses, coronary disease and possible echinococcal cyst of the liver. The second death was partly due to hæmorrhage eight days after peritoneoscopy with liver biopsy for extensive metastatic carcinoma. The third death occurred three weeks after perforation of the large bowel in a patient who was severely ill and was proved by peritoneoscopy to be suffering from advanced tuberculous peritonitis. When the advantages that may be gained by skilfully performed peritoneoscopy are weighed against the deaths of three patients who were so acutely ill as these, there would appear to be little ground for objection to its use on the score of risk. In 177 of the 331 cases in which liver disease was suspected, malignant conditions of the liver were found, in 83 non-malignant conditions were discovered, and in the remaining 71 cases the appearances were essentially normal. Of the 177 malignant conditions, 170 were cases of metastatic carcinoma; five were primary carcinomata. Of the 73 non-malignant conditions, 83 were cases of cirrhosis. Benedict states that in regard to the liver the question to be decided is often whether any disease of the liver is present, and if so, whether the condition is malignant disease, cirrhosis or something unsuspected. Benedict's paper contains many case histories which should be studied by those specially interested in the subject. For others we intend to refer to some of his observations showing the advantages of the method. Peritoneoscopy was performed in 95 cases of carcinoma of the stomach; in 75 no metastases were found and in twenty cases metastases were seen. The result was that laparotomy was avoided in twenty cases. On the other hand, during a five-year period 263 patients with cancer of the stomach were submitted to exploratory operation without previous peritoneoscopy. In 39 of these cases, or 15%, the condition was found to be inoperable because of liver metastasis.

[1] *The New England Journal of Medicine*, February 3, 1944.

Benedict points out that these 39 patients might have avoided the ordeal of operation if peritoneoscopy had been used. He quotes a case to show that a patient's condition may appear to be inoperable, but favourable findings on peritoneoscopy may lead to successful resection. In another case peritoneoscopy revealed an unsuspected cirrhosis of the liver in a patient known to have carcinoma of the stomach, and laparotomy was avoided. In yet another case the presumptive clinical diagnosis was pulmonary tuberculosis and splenomegaly of undetermined cause. Peritoneoscopy showed that the whole process was due to sarcoid. The five histories of primary carcinoma of the liver are given in detail, and it is shown that in at least three of the cases diagnosis was made possible by peritoneoscopy.

Benedict does not mention the use of any supplementary instrument such as that which Hamilton has described; he would not as a general rule be likely to need one in dealing with an organ such as the liver. The use of Hamilton's "manipulator" would, it must be concluded, call for considerable dexterity and great discrimination. Hamilton reported that he had used his instrument thirty-one times in 125 peritoneoscopies without the occurrence of accident and with gratifying results. By its means he was able to extend the use of peritoneoscopy. He was able to discover penetration of the bowel in cases of gunshot and stab wounds of the abdomen and to undertake immediate laparotomy. In several instances he claimed that he was able to decide that laparotomy was not necessary. He found it useful in the differentiation of appendicitis and salpingitis and of appendicitis and cholecystitis. He used it to investigate non-penetrating injuries when internal trauma was suspected, and also to differentiate bleeding peptic ulcer from ruptured œsophageal varix. Those who contemplate the use of this method (and its wider use certainly seems justified) should remember the smallness of the incision under local anæsthesia, the lessened risk and discomfort for the patient and shortness of the necessary stay in hospital.

ACUTE GENERALIZED MILIARY TUBERCULOSIS.

SOME interesting observations have been "presented for consideration" by Oscar Auerbach[1] on the subject of acute generalized miliary tuberculosis. Reference to text-books on pathology will show the general acceptance of the view that dissemination of the infection takes place by the blood stream after the direct discharge into it of the contents of a tuberculous focus. For example, adhesions may form between a caseous gland and a large vein. A tuberculous lesion in the thoracic duct, it is held, may undergo ulceration and discharge its contents into the lumen to find their way to the venous circulation. Auerbach holds that the role of active extrapulmonary tuberculous processes in the production of the dissemination has been underestimated and that too much stress has been laid on the search for vascular foci. He reports that among 1,656 consecutive autopsies on tuberculous persons, performed over a period of eight and a half years, there were 297 cases, or 17·9%, of acute generalized miliary tuberculosis. Of the 297 cases 191 manifested varying degrees of chronic pulmonary tuberculosis in combination with the miliary dissemination; in 106 cases a generalized miliary seeding existed alone. Auerbach recalls that Orth found more than half of his thirty cases of acute generalized miliary tuberculosis were associated with chronic pulmonary tuberculosis; he adds that Orth is the only writer whose findings in this respect coincide with his own. Weigert and all other authors who have discussed this relationship express their conviction that the presence of chronic pulmonary tuberculosis excludes the development of acute miliary tuberculosis. Auerbach believes that the discrepancy between the findings of these authors and his own arises from the fact that they have interpreted the miliary foci found in lungs in conjunction with chronic pulmonary tuberculosis as representing bronchogenic spread from these chronic pulmonary lesions, and have interpreted the miliary foci in other organs as terminal hæmatogenous disseminations from the lungs.

Auerbach discusses shortly the views that have been held regarding pathogenesis. Laennec, he states, was the first to point out that older soft caseous foci could lead to extensive dissemination throughout other organs. Buhl went further and expressed the opinion that in the majority of cases of acute generalized miliary tuberculosis there were older caseous foci in the body. Huguenin believed that the caseous mass could either erode and enter a small vein or invade the lymphatics. Weigert stated that since the caseous focus was not frequently found, its occasional presence in cases of acute miliary tuberculosis was not sufficient evidence for belief in it as the source of the dissemination. Weigert pointed to the infrequent occurrence of acute generalized miliary tuberculosis in conjunction with well-developed phthisis. He argued that as there were in phthisis caseous processes and softened foci as well, and as these were apparently insufficient for the development of acute miliary tuberculosis, there must be other factors which allowed the entry of masses of tubercle bacilli into the blood stream. In his studies Weigert came across a case of acute generalized miliary tuberculosis in which a large caseous lymph node extended into the right innominate vein. He subsequently found other "venous tubercles" in cases of the disease. Weigert therefore contended that the vascular tubercle progressed from a tuberculous focus, that when extension occurred in the vessel wall the intima was destroyed and rupture into the blood stream occurred. This view was accepted by many workers. Auerbach gives a short account of the views of other authors who have held variations of the view put forward by Weigert. He rejects them all. His studies do not allow him to accept any of the views of pathogenesis which are based on the assumption of the rupture of a tuberculous focus into the blood stream. It is his opinion that the development of acute generalized miliary tuberculosis is the result of the drainage of tubercle bacilli from an active extrapulmonary tuberculous focus or a primary focus into the lymphatic system which in turn empties into the venous system. He points out that Buhl, already mentioned, held that lymphatic drainage from a caseous focus might occur; he also refers to Ponfick, who found that in the majority of his cases of acute generalized miliary tuberculosis he could demonstrate small tubercle-like nodules within the intima of the thoracic duct. Auerbach has found in every one of his cases an acute tuberculous process in one or more organ systems. This process he has found to be almost invariably characterized by caseous tuberculosis of the regional lymph nodes. Lymph node involvement indicates extensive lymphatic drainage of tubercle bacilli from neighbouring organ tuberculosis—the process being analogous to that seen in any infection. The migration of tubercle bacilli into lymph nodes has been demonstrated by bacterial stains. "The tubercle bacilli are carried in the lymph to the venous circulation, and a generalized blood stream dissemination takes place with the development of miliary foci in the various organs of the body." Auerbach points out that lymphatic drainage with subsequent hæmatogenous dissemination is observed in cases of chronic pulmonary tuberculosis, and that the dissemination occurs most frequently into the spleen and kidney, but sometimes also into the adrenals and other organs including the brain. This hæmatogenous dissemination was observed by him in 33·2% of the cases in which chronic pulmonary tuberculosis was present without associated extrapulmonary foci.

On the clinical side it may be useful to draw attention to Auerbach's observation, as a result of radiological study, that a hæmatogenous dissemination of tubercle bacilli sufficient to produce generalized miliary foci is not necessarily an incurable condition. He has observed the almost invariable occurrence of multiple "seedings", some or all of which may resolve.

Auerbach's paper concludes with no less than fifty-one references, almost every one of which is to German literature. The subject seems to have been neglected lately in English and American journals. Auerbach's paper is well worth the consideration that he asks for it.

[1] *The American Journal of Pathology*, January, 1944.

Reviews.

MEDICINE AND SOCIOLOGY.

"MEDICINE AND THE SOCIAL ORDER", by Dr. E. P. Dark, is a remarkable book; its second impression has appeared.[1] From a basis of three articles, "Medicine and the Social Order", "Property and Health", "Some Medical Aspects of Crime", all of which have appeared in THE MEDICAL JOURNAL OF AUSTRALIA, the author has wandered widely afield into the causes of war, the problems of peace, education, and medicine in Russia. He deals with his subjects in a broad-minded way, gathers his facts from the far corners of the earth and in Australia, and then delivers his verdict plainly, courageously and clearly. One of the chief merits of the book is that it compels attention even from those who may differ from its "leftish" viewpoint. Dr. Dark pillories the old social order, but in doing so asks those of the "right" to state their case. The map is before our eyes—we must travel to the right or the left. The reader will not be long in doubt before he realizes why the author passes to the left.

Dr. Dark believes that "health is bound up with the political and economic structure of society, so that the medical profession as a whole is inevitably interested in politics and economics, and should speak with authority on all political and sociological questions which have any bearing upon health". In support of this contention he draws attention to the investigations on the relationship between diet, economic status and health. The data are terrible in their implications. That tuberculosis should be so much more common in unskilled labourers than in the more highly endowed, that privileged, well-fed children should be 3·8 inches taller at seventeen than employed children, are some of the pointers to an inescapable conclusion. Medicine and the economic system are closely linked. We must not sit idly to contemplate without action the cycles of depression, the burning of foodstuffs, the social decay which followed in the wake of the last war. Dr. Dark states: "I am convinced that it is time the medical profession became the leaders of the people in all matters that concern the public health."

In the discussion on property, there is mention of the Stockton-on-Tees experiment. Although better housing facilities were given, the result was an increased morbidity. The extra money spent on rent reduced the food allowance, resulting in a 50% increase in the death rate.

Concerning the slum evil, the reader will learn that Australia is not blameless. Already our slums are creating a serious problem in health which can be solved only by a drastic revision of our social outlook. Our author believes that:

> The Government must be told in a way it cannot ignore, that its most fundamental need is for fit men, and that it cannot have fit men until it can assure sufficient food, decent shelter, and work for everyone. This spreading of essential knowledge can be effectively done only by the medical profession through its official associations.

The chapter on "Some Medical Aspects of Crime" is outstanding. Attention is drawn to the dreary monotony of prison life and prison fare, the aim of which is punitive rather than remedial. Dr. Dark points out that in Russia there have been outstanding improvements in the penal code. Its aim is not to deter others, but to re-create citizens and conduct research in criminality.

At first sight the relationship between medicine and the causes of war is not apparent. If, however, it can be shown that economic factors are largely the basis for all war, we are faced with our need to meddle in sociology. Dr. Dark's evidence is intriguing and damning. He considers the Peloponnesian War, the Punic Wars, the Crusades and contemporary history.

It is essential to define the nature of "the State". He uses Laski's thesis that it is merely the coercive power wielded by the dominant group in a country and used primarily to prevent any change that might imperil its power or its profits. In the author's opinion, when capitalism is dominant, the results are predetermined.

> Capitalism, once fully established, has proved to mean over-production and under-consumption; it can produce abundantly, but it cannot get its products distributed. When the home market was glutted the position was temporarily eased by export of goods, capital goods and capital. To get and control the markets for these, the more backward parts of the world were divided up, and monopoly took the place of competition. When this stage is finished and all the world is parcelled out, the inevitable drive to war is clearly manifest, for capitalism must still expand, and it can only do so at the expense of a rival. At the same time the great monopolies must have absolute control of the State to regulate industry to their own advantage, and to reduce the workers once more to serfdom. Here is the inevitable evolutionary chain—competitive capitalism, monopoly, imperialism, Fascism, war.

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CARRIERS AND ENTERIC FEVER.

WHEN the term "carrier" is used in reference to a particular infectious disease it indicates a "person who has within himself the germs or other infective virus of that infectious disease, but has no other sign or symptom thereof". In enteric fever the ease with which typhoid bacilli in the alimentary canal of a carrier may infect a healthy person needs no emphasis; and the treatment of a patient recovering from the disease will not be complete until it has been determined that he or she is not a carrier. In the account given a week or two ago in these pages of the outbreak of enteric fever that occurred at Moorabbin, Victoria, in March, 1943, the subject of carriers was mentioned. It should, however, be pursued further for several reasons. One reason is contained in the following statement by Dr. F. V. G. Scholes: "If we assume that there still exist one or more undetected persistent carriers, as well as those known and under surveillance, there must be some uneasiness about the possibility of further large or small outbreaks in Melbourne during the next year or two." Again, apart from the general importance of the subject, there is considerable interest in the way in which the carrier question was handled in the Moorabbin epidemic.

In the special report on chronic enteric carriers and their treatment, prepared for the Medical Research Council of Great Britain in 1933 by C. H. Browning and others (see THE MEDICAL JOURNAL OF AUSTRALIA, June 10, 1933) a clear exposition is given. In the first place the available evidence points to the fact that the typhoid bacillus has a very limited capacity for survival in nature apart from the human host, and chronic carriers are therefore one of the chief sources from which fresh epidemics arise. The second consideration of importance is that the spontaneous cure of a chronic carrier practically never occurs. Browning described temporary and permanent or chronic carriers. In convalescence from enteric fever many persons excrete the specific organisms for short periods; these are the temporary carriers. It is of great importance, however, to note that excretion of specific organisms in the fæces may set in some time, even several months, after convalescence. This lateness of onset occurs oftener with enteric bacilluria. Most of those who continue to excrete enteric bacilli for six months after the acute attack will continue to do so. Those who continue to excrete the bacilli after a year will not become cured spontaneously and are to be regarded as permanent or chronic carriers. From another point of view carriers may excrete the organisms from the intestinal tract or in the urine; rarely organisms may be excreted from other sites such as the sinuses. Those who excrete bacilli from the intestinal tract may be biliary carriers or true intestinal carriers or both. That it is important to recognize the true intestinal carrier is sometimes obvious during treatment; it is not unlikely that some of the failures to obtain cures by cholecystectomy may be due to the fact that the patients concerned are true intestinal and not biliary carriers. Browning discusses at some length the problem set by biliary carriers and makes reference to the pathology. The most marked histological changes observed in all gall-bladders consisted of localized and diffuse collections of cells in the mucosa, and these are mainly plasma cells and lymphocytes with scanty eosinophile cells. He refers to cases in which clumps of bacilli are seen on the mucosa of the gall-bladder, but none are found in its substance. He therefore finds considerable support for the opinion of Garbat that in such cases the carrier state is maintained by the gall-bladder acting as "a test-tube containing the bile medium in which the typhoid bacteria propagate without affecting the gall-bladder itself". It is of interest to note that the association of gall-stones with the enteric carrier state is well established. Browning points out that it is seldom possible to decide in any particular instance whether the inflammatory change in the wall of the gall-bladder with the formation of calculi has been initiated by the enteric infection or whether it was on an abnormal mucosa, with or without the presence of gall-stones, that the typhoid bacillus could establish and maintain itself. Even when *Bacillus typhosus* or *Eberthella typhi*, to give the organism its modern name, is found in the centre of calculi in the gall-bladder, this is no indication that the bacterial infection preceded the gall-stone formation, for it is known that the organisms may readily penetrate cracks in stones. In regard to urinary carriers, Browning points out that when the bacilli persist in the urine it is the rule to find in the urinary tract some pathological condition which is accompanied by pyuria. The nidus of infection is situated most commonly in the kidney or the kidney pelvis. In these circumstances surgical treatment is indicated.

Since the carrier state causes the spread of the disease, and since the carrier state is dependent on the presence of lesions such as those mentioned, it would appear quite simple to arrange matters so that no carrier should pass out of control until he had ceased to be dangerous to other people, in other words, until he was no longer a carrier. But this ideal procedure cannot always be carried out. It could not be adopted at Moorabbin and the health authorities displayed both wisdom and common sense in their handling of the situation. Dr. Merrillees states his conviction that, although the carrier state is potentially dangerous, it is not actually dangerous unless (*a*) the carrier has habits which make the infection of other persons likely, and (*b*) unless an opportunity arises for the handing on of the infection. At Moorabbin carriers could not be isolated indefinitely because the general hospitals and the infectious diseases hospital needed beds for other patients. It was decided that the "best" of the

carriers could be discharged to their homes, temporarily at least, to see if their abnormal condition would clear up. Those allowed to go home were those who (*a*) were intelligent and cooperative, (*b*) did not handle food supplies, (*c*) had a private home to go to where conditions were suitable, (*d*) were habitually clean. In regard to the homes, four requirements had to be met: the house had to be sanitary, not crowded and not associated with any public food supply; the house had to be a single private house, not divided into flats and not a boarding house; the drainage and sanitary services had to be effective; all the inmates had to submit to supervision and cooperate with the patient and the health authority. Obviously each carrier had to be considered as a special problem. Very few adults would be detained if they had suitable homes to go to; but infancy, senility and subnormal mentality needed special consideration. Where one man, clean, intelligent and anxious to learn, was released without question, another, somewhat submental, willing to please, but unable to grasp the meaning of the ritual of cleanliness, was persuaded to undergo cholecystectomy with satisfactory results. Carriers released under the conditions mentioned were regarded as released on probation; they are being kept under surveillance and frequent examinations of urine and fæces are made. "Should these persistently show positive the position will be reviewed in say three to six months and other treatment advised or not according to the opinion of consultants."

Expertly though this outbreak was handled, there still remains what Dr. Merrillees calls "grave danger" from missed cases. He points out that though the outbreak has shown the great need for examination of convalescents in regard to their carrier state, it has also shown that the disease can exist in all degrees of severity from an overwhelming toxic disaster down to a trivial, barely noticed indisposition. "No matter how careful we may be in respect to detecting and educating chronic carriers among the convalescents, there is an absolute certainty that some carriers will escape discovery because they never have been patients." Only a superficial view has been taken here of some of the aspects of an enormous subject; the main thing is that practitioners should never allow the idea of a disease greatly lessened in incidence to lead to any complacency about its sporadic appearance. Perhaps one of the most significant statements in Dr. Merrillees's excellent report is contained in the following words: "This outbreak shows we have signally failed so far; we owe this outbreak to a previously undetected carrier working on a dairy farm and with not even the rudiments of hygiene." In the ideally hygienic state of the future no one will be allowed to handle food for distribution until he or she has been certified as free from communicable disease and addicted to clean habits.

Current Comment.

PERITONEOSCOPY.

THOUGH the method of examination known as peritoneoscopy has been known for many years and reliable surgeons have claimed that it has a sphere of usefulness, it has never been widely used. Reports have appeared in the literature from time to time, and in the last year or two these have been more numerous. It was as long ago as 1901 that Kelling first described the method of examining the peritoneal cavity by means of a cystoscope introduced through a small incision in the abdominal wall. Writing in the *British Medical Journal* of August 8, 1925, A. Rendle Short described briefly the advantages of the method by which air is introduced into the peritoneal cavity through a tiny incision made under local anæsthesia and a cystoscope is introduced for the inspection of the viscera. He stated that the method (he used the term cœlioscopy) did not and could not replace exploratory laparotomy when the cause of a pathological condition might have to be sought which did not lie on the surface of the abdominal viscera. These he thought would include the majority of lesions. The method, he declared, was principally valuable for what was definitely seen and not for what was apparently absent. Short remarked that it was like every other surgical procedure in that a little trouble had to be taken to learn how to obtain a good view, but it was no more difficult than cystoscopy. R. M. Walker and P. L. Playfair, writing in *The Lancet* of February 7, 1942, make much the same kind of remark. At first a cystoscope was used for this work, but a special peritoneoscope has been evolved and is generally used. It is inserted after a pneumoperitoneum has been produced with air introduced through a special trocar. In addition J. E. Hamilton has described in *Surgery, Gynecology and Obstetrics* of February, 1942, a new "manipulator" which may be used in conjunction with the peritoneoscope. It consists of a blunt-tipped rod, the manipulator proper, and an insulated endothermy electrode, both of which fit into a cannula such as is used with a trocar for paracentesis. The trocar and cannula are introduced through a separate stab incision under vision with the peritoneoscope; the trocar is then removed and the manipulator or the electrode may be inserted. The manipulator is used to push on one side structures that obstruct the view through the peritoneoscope.

E. B. Benedict, writing from the Massachusetts General Hospital, has recently reported experiences with peritoneoscopy in liver disease.[1] In a series of 435 peritoneoscopy operations the question of liver disease arose in 331 or 70%. Among the 435 cases there were three in which death was probably caused by the procedure. One death resulted from heart failure and was that of a patient who suffered from multiple lung abscesses, coronary disease and possible echinococcal cyst of the liver. The second death was partly due to hæmorrhage eight days after peritoneoscopy with liver biopsy for extensive metastatic carcinoma. The third death occurred three weeks after perforation of the large bowel in a patient who was severely ill and was proved by peritoneoscopy to be suffering from advanced tuberculous peritonitis. When the advantages that may be gained by skilfully performed peritoneoscopy are weighed against the deaths of three patients who were so acutely ill as these, there would appear to be little ground for objection to its use on the score of risk. In 177 of the 331 cases in which liver disease was suspected, malignant conditions of the liver were found, in 83 non-malignant conditions were discovered, and in the remaining 71 cases the appearances were essentially normal. Of the 177 malignant conditions, 170 were cases of metastatic carcinoma; five were primary carcinomata. Of the 73 non-malignant conditions, 83 were cases of cirrhosis. Benedict states that in regard to the liver the question to be decided is often whether any disease of the liver is present, and if so, whether the condition is malignant disease, cirrhosis or something unsuspected. Benedict's paper contains many case histories which should be studied by those specially interested in the subject. For others we intend to refer to some of his observations showing the advantages of the method. Peritoneoscopy was performed in 95 cases of carcinoma of the stomach; in 75 no metastases were found and in twenty cases metastases were seen. The result was that laparotomy was avoided in twenty cases. On the other hand, during a five-year period 263 patients with cancer of the stomach were submitted to exploratory operation without previous peritoneoscopy. In 39 of these cases, or 15%, the condition was found to be inoperable because of liver metastasis.

[1] *The New England Journal of Medicine*, February 3, 1944.

Benedict points out that these 39 patients might have avoided the ordeal of operation if peritoneoscopy had been used. He quotes a case to show that a patient's condition may appear to be inoperable, but favourable findings on peritoneoscopy may lead to successful resection. In another case peritoneoscopy revealed an unsuspected cirrhosis of the liver in a patient known to have carcinoma of the stomach, and laparotomy was avoided. In yet another case the presumptive clinical diagnosis was pulmonary tuberculosis and splenomegaly of undetermined cause. Peritoneoscopy showed that the whole process was due to sarcoid. The five histories of primary carcinoma of the liver are given in detail, and it is shown that in at least three of the cases diagnosis was made possible by peritoneoscopy.

Benedict does not mention the use of any supplementary instrument such as that which Hamilton has described; he would not as a general rule be likely to need one in dealing with an organ such as the liver. The use of Hamilton's "manipulator" would, it must be concluded, call for considerable dexterity and great discrimination. Hamilton reported that he had used his instrument thirty-one times in 125 peritoneoscopies without the occurrence of accident and with gratifying results. By its means he was able to extend the use of peritoneoscopy. He was able to discover penetration of the bowel in cases of gunshot and stab wounds of the abdomen and to undertake immediate laparotomy. In several instances he claimed that he was able to decide that laparotomy was not necessary. He found it useful in the differentiation of appendicitis and salpingitis and of appendicitis and cholecystitis. He used it to investigate non-penetrating injuries when internal trauma was suspected, and also to differentiate bleeding peptic ulcer from ruptured œsophageal varix. Those who contemplate the use of this method (and its wider use certainly seems justified) should remember the smallness of the incision under local anæsthesia, the lessened risk and discomfort for the patient and shortness of the necessary stay in hospital.

ACUTE GENERALIZED MILIARY TUBERCULOSIS.

SOME interesting observations have been "presented for consideration" by Oscar Auerbach[1] on the subject of acute generalized miliary tuberculosis. Reference to text-books on pathology will show the general acceptance of the view that dissemination of the infection takes place by the blood stream after the direct discharge into it of the contents of a tuberculous focus. For example, adhesions may form between a caseous gland and a large vein. A tuberculous lesion in the thoracic duct, it is held, may undergo ulceration and discharge its contents into the lumen to find their way to the venous circulation. Auerbach holds that the role of active extrapulmonary tuberculous processes in the production of the dissemination has been underestimated and that too much stress has been laid on the search for vascular foci. He reports that among 1,656 consecutive autopsies on tuberculous persons, performed over a period of eight and a half years, there were 297 cases, or 17·9%, of acute generalized miliary tuberculosis. Of the 297 cases 191 manifested varying degrees of chronic pulmonary tuberculosis in combination with the miliary dissemination; in 106 cases a generalized miliary seeding existed alone. Auerbach recalls that Orth found more than half of his thirty cases of acute generalized miliary tuberculosis were associated with chronic pulmonary tuberculosis; he adds that Orth is the only writer whose findings in this respect coincide with his own. Weigert and all other authors who have discussed this relationship express their conviction that the presence of chronic pulmonary tuberculosis excludes the development of acute miliary tuberculosis. Auerbach believes that the discrepancy between the findings of these authors and his own arises from the fact that they have interpreted the miliary foci found in lungs in conjunction with chronic pulmonary tuberculosis as representing bronchogenic spread from these chronic pulmonary lesions, and have interpreted the miliary foci in other organs as terminal hæmatogenous disseminations from the lungs.

Auerbach discusses shortly the views that have been held regarding pathogenesis. Laennec, he states, was the first to point out that older soft caseous foci could lead to extensive dissemination throughout other organs. Buhl went further and expressed the opinion that in the majority of cases of acute generalized miliary tuberculosis there were older caseous foci in the body. Huguenin believed that the caseous mass could either erode and enter a small vein or invade the lymphatics. Weigert stated that since the caseous focus was not frequently found, its occasional presence in cases of acute miliary tuberculosis was not sufficient evidence for belief in it as the source of the dissemination. Weigert pointed to the infrequent occurrence of acute generalized miliary tuberculosis in conjunction with well-developed phthisis. He argued that as there were in phthisis caseous processes and softened foci as well, and as these were apparently insufficient for the development of acute miliary tuberculosis, there must be other factors which allowed the entry of masses of tubercle bacilli into the blood stream. In his studies Weigert came across a case of acute generalized miliary tuberculosis in which a large caseous lymph node extended into the right innominate vein. He subsequently found other "venous tubercles" in cases of the disease. Weigert therefore contended that the vascular tubercle progressed from a tuberculous focus, that when extension occurred in the vessel wall the intima was destroyed and rupture into the blood stream occurred. This view was accepted by many workers. Auerbach gives a short account of the views of other authors who have held variations of the view put forward by Weigert. He rejects them all. His studies do not allow him to accept any of the views of pathogenesis which are based on the assumption of the rupture of a tuberculous focus into the blood stream. It is his opinion that the development of acute generalized miliary tuberculosis is the result of the drainage of tubercle bacilli from an active extrapulmonary tuberculous focus or a primary focus into the lymphatic system which in turn empties into the venous system. He points out that Buhl, already mentioned, held that lymphatic drainage from a caseous focus might occur; he also refers to Ponfick, who found that in the majority of his cases of acute generalized miliary tuberculosis he could demonstrate small tubercle-like nodules within the intima of the thoracic duct. Auerbach has found in every one of his cases an acute tuberculous process in one or more organ systems. This process he has found to be almost invariably characterized by caseous tuberculosis of the regional lymph nodes. Lymph node involvement indicates extensive lymphatic drainage of tubercle bacilli from neighbouring organ tuberculosis—the process being analogous to that seen in any infection. The migration of tubercle bacilli into lymph nodes has been demonstrated by bacterial stains. "The tubercle bacilli are carried in the lymph to the venous circulation, and a generalized blood stream dissemination takes place with the development of miliary foci in the various organs of the body." Auerbach points out that lymphatic drainage with subsequent hæmatogenous dissemination is observed in cases of chronic pulmonary tuberculosis, and that the dissemination occurs most frequently into the spleen and kidney, but sometimes also into the adrenals and other organs including the brain. This hæmatogenous dissemination was observed by him in 33·2% of the cases in which chronic pulmonary tuberculosis was present without associated extrapulmonary foci.

On the clinical side it may be useful to draw attention to Auerbach's observation, as a result of radiological study, that a hæmatogenous dissemination of tubercle bacilli sufficient to produce generalized miliary foci is not necessarily an incurable condition. He has observed the almost invariable occurrence of multiple "seedings", some or all of which may resolve.

Auerbach's paper concludes with no less than fifty-one references, almost every one of which is to German literature. The subject seems to have been neglected lately in English and American journals. Auerbach's paper is well worth the consideration that he asks for it.

[1] *The American Journal of Pathology*, January, 1944.

Abstracts from Medical Literature.

PHYSIOLOGY.

Severe Anoxic Anoxia with Recovery.

R. L. WARD AND O. C. OLSON (*The Journal of Aviation Medicine*, December, 1943) describe the most severe case of anoxic anoxia with recovery that has ever been reported. The patient was a member of the crew of a bomber flying at an altitude of 25,000 feet. It became necessary for the patient to leave his post and go to the bomb bay. He attached his oxygen inlet hose to a "walk around" oxygen bottle and carried out his task. Fifty-five minutes afterwards a search disclosed him, unconscious, apnœic and markedly cyanotic with ice in mouth and nostrils. The log of the plane allowed an accurate estimate of the oxygen pressures to which he had been exposed. The patient was unconscious for eight hours and semicomatose for an additional eleven hours. Neurological examinations during the first twenty hours revealed diminished to absent deep and superficial reflexes, and a bilateral Babinski reflex. There was also an increased spinal fluid pressure with projectile vomiting. The patient had no subjective symptoms, such as headaches or dizziness, at any time. Psychological changes lasted about six days and subsequently entirely cleared up, leaving no apparent intellectual defects or personality changes. The hyperpyrexia, which was an early, acute phenomenon, can probably be attributed to a temporary disturbance of the heat centre. It has been shown experimentally in animals that a reduction of partial pressure of oxygen has an effect on the body temperature. In many cases of cerebral concussion, as well as in some cases of cerebral anoxia, elevations of body temperature are present. The restlessness, spasticity and mild clonic movements as seen in the early stages of this patient's history are similar to those demonstrated in patients undergoing short periods of severe anoxia. The psychological changes were most interesting, and gave rise to much speculation. It was the belief of most observers initially that there would be some remaining permanent intellectual defects and personality changes. None are demonstrable at present. Whether or not such changes will become evident in the future only time will tell. Although this is an isolated case and there is the factor of individual susceptibility, it brings up the question as to whether the prognosis in patients suffering from prolonged anoxia is as dire as was formerly assumed.

The Origin of High Altitude Pain.

D. E. ROSENBLOOM (*Bulletin of Experimental Biology and Medicine*, Moscow, Number 6, 1943) surveys the symptoms and ætiology of high altitude pain in the joints as commonly seen in air crews. Statistical material collected in a great number of cases shows that the frequency of articular pain increases with the increase of altitude: 5% to 10% at 8,000 metres, 20% to 30% at 10,000 metres, 40% at 12,000 metres. The different explanations given the phenomenon, such as oxygen hunger or increase of previously existing rheumatic pains, do not conform with experimental results (obtained in low pressure chambers) or with carefully collected histories in which any previous rheumatic symptoms are excluded. According to the view expressed long ago by Holden, Daman and Boycott, this kind of pain would be due to nitrogen set free in gas form from tissues where it is normally dissolved. This syndrome would be similar to that observed in deep-sea divers or caisson workers when they come rapidly to the surface. Experiments conducted on pilots and laboratory animals have demonstrated the identity of the origin of the caisson disease and high altitude pain. The prevention of the pain can be obtained by depriving the tissues of nitrogen through prolonged breathing of oxygen. This should be done through hermetically sealed masks to avoid introduction of atmospheric nitrogen together with the oxygen.

Increased Red Blood Cell Fragility after Fat Ingestion.

J. LONGINI AND V. JOHNSON (*The American Journal of Physiology*, December, 1943) report some experiments made on dogs which demonstrate the presence in the lipæmic serum of one or more agents which act directly on red blood cells to increase their fragility. Neutral fat is not this agent. Previous work indicates that free fatty acids and soaps, reaching the blood stream by way of the lymphatics, are probably responsible for the effect. Ingestion of carbohydrates and proteins does not increase red cell fragility. On the contrary, meals rich in these substances produce serum which may actually protect red cells from water hæmolysis. These results, when considered with the finding of increased red cell destruction *in vivo*, on a high fat diet, indicate that fat ingestion probably is an important mechanism in the physiological destruction of red cells in dogs. These experiments suggest further that the mechanism of the demonstrated in-vivo red cell destruction by fat ingestion is immediate and direct and may not involve any relatively long metabolic process. It is suggested that those physiological mechanisms which delay the absorption of fat and its entrance into the blood stream, are probably significant in the prevention of anæmia.

Persistence of Heat Acclimatization in Man.

A. HENSCHEL, H. L. TAYLOR AND A. KEYS (*The American Journal of Physiology*, December, 1943) describe some experiments designed to test the persistence of heat acclimatization in man. The performance of muscular work in dry heat was studied in twenty-four normal young men. The diet, environment and work routine were carefully controlled during the stay in the heat. Comparisons were made between the performances in the heat on two occasions of two days each separated by from one to four weeks of cold weather. Work pulse rates, rectal temperatures and, to a lesser degree, vasomotor stability tests showed that heat acclimatization persists during at least three weeks of cold weather. The benefit conferred by this acclimatization is important for this time, but the advantage decreases from week to week. The authors mention that in the popular Press there have been statements that German troops destined for hot-weather service in the African deserts were "conditioned" in heated buildings in Germany. From the present results it is clear that such preparation might be of real value even though as long as several weeks elapsed between acclimatization in Germany and the operation in Africa.

Environmental Temperatures and the Survival of Dogs after Severe Bleeding.

R. A. CLEGHORN (*The Canadian Medical Association Journal*, November, 1943) reports some experiments made on dogs to support the statement made by Bazett, that the use of heat in cases of shock in which the blood volume is seriously reduced, is wrong in principle and bad in practice. The influence of different environmental temperatures on the survival of critically bled dogs was investigated. The mortality at four different temperatures was as follows: 18% at 72° F., 38% at 52° F., 45% at 85° F., and 93% at 95° F. The higher mortality in the dogs at 52° F. compared with those at 72° F. is ascribed to the increased oxygen requirement. The higher mortality in the dogs at 85° F. compared to those at 72° F. is attributed to the deleterious influence of vasodilatation caused by this degree of warming. The insensible water loss in this group was not a factor, as it was the same at both temperatures. The higher mortality in the dogs at 95° F. compared with those at 72° F. is ascribed to vasodilatation and to increase in insensible water loss associated with the panting which occurred at this temperature. To another group of dogs exposed to 95° F. an amount of water equivalent to the increase in the insensible water loss was given. The mortality in this group was reduced to approximately that seen in the dogs exposed to 85° F. The difference in the responses of dogs and man to elevation of the environmental temperature are briefly discussed and the reasons are given why patients who are suffering from secondary shock or severe acute hæmorrhage should not be heated.

Some Effects of Low Barometric Pressures on Kidney Function in the White Rat.

H. SILVETTE (*The American Journal of Physiology*, December, 1943) discusses the literature concerning the effect of anoxia on kidney function in various animals. He then states that results obtained in his laboratory indicate that rats respond in a manner corresponding to the human response rather than to that of dogs. Exposure to sufficiently low barometric pressures for three-hour periods invariably led to polyuria in the white rat. After exposure to 15,000 feet altitude equivalent the urine output was increased 150% and after 25,000 feet exposure 300% over the control (0 foot) levels. The high altitude polyuria was further increased by cold (10° C.), but decreased by heat (30° C.). Phenolsulphonephthalein excretion was not significantly affected during exposure to 15,000 feet altitude equivalent. In contrast to the polyuria with normal phenolsulphonephthalein excretion

observed at high altitudes, anæmic anoxia due to carbon monoxide poisoning resulted in decreased urine and dye excretion. The greater the water-load, the greater was the absolute excretion of urine at 15,000 feet, but the less the relative increase in urinary output over the control (0 foot) levels. Repeated three-hour daily exposures of white rats to simulated altitudes of 15,000 or 25,000 feet had no significant effect on their polyuric response, which was well sustained throughout the twenty-seven-day experiments. At the end of this time, when tested at 0 foot once more, the urinary excretion was normal or returned to normal within a few days. Relative kidney weights of these animals indicated a slight degree of renal hypertrophy after the period of 15,000 feet exposure, a more marked degree of hypertrophy after 25,000 feet exposure, but an apparent atrophy after a one-month recovery period at room pressure. The histological picture of the kidneys of animals after long intermittent exposures to 15,000 feet was normal, except for marked hyperæmia, but at 25,000 feet early slight pathological changes occurred. The polyuria of high altitudes may be inhibited or reversed by the injection of post-pituitary extract.

BIOCHEMISTRY.

Water Metabolism.

THE water metabolism of hypertensive rats has been investigated by K. A. Oster and O. Martinez (*The Journal of Experimental Medicine*, December, 1943), who followed the water intake. Rats made hypertensive by renal ischæmia increased their water consumption by 75% over the preoperative level. Polyuria was associated with this polydipsia and the independence of these occurrences from a number of other factors was demonstrated. It was found that the presence of a normal kidney exerted a compensatory influence which might mask either hypertension or polyuria or both. The appearance or exacerbation of the changes upon removal of the normal kidney, on the one hand, and the elimination or mitigation of the symptoms upon the removal of the ischæmic kidney, on the other, support the view that the changes observed cannot have been due to passive elimination of the kidney tissue by ischæmia, but to active malfunction of the renal, and especially the tubular, mechanism upon withdrawal of oxygen. The view is put forward that polyuria is a primary sequel of ischæmia rather than secondary to the intrarenal and extrarenal effects of hypertension. A number of concomitant observations are in harmony with this hypothesis.

Shock.

J. A. RUSSELL *et alii* have investigated the role of the peripheral tissues in the metabolism of protein and carbohydrate during hæmorrhage shock in the rat (*The Journal of Experimental Medicine*, January, 1944). The changes in the blood levels of amino nitrogen, glucose, lactate and pyruvate were compared in eviscerated rats and eviscerated rats subjected to hæmorrhage, in order to establish the role of the peripheral tissues in the blood changes during shock. It was found that the blood amino acids accumulate at a more rapid rate in the bled liverless rats than in the control liverless animals. The blood sugar level falls rapidly in the liverless rat after hæmorrhage, both in animals with intact suprarenal glands and those with enucleated suprarenal medullæ. The blood lactate and pyruvate levels rise slowly in the liverless rat, but maintain a constant relation to each other, except terminally when convulsions occur. In the bled liverless rat both the lactate and pyruvate contents increase much more rapidly than in the control liverless rat, and the lactate:pyruvate ratio also increases. These data are interpreted to mean that a decrease in liver function during hæmorrhage shock serves to make apparent a considerable increase in peripheral protein catabolism and accentuates the effects of an increased carbohydrate utilization by the periphery. The lactate and pyruvate changes are determined chiefly by anoxia of the peripheral tissue and probably indicate an increasing predominance of anaerobic over aerobic metabolism of carbohydrate in muscle. The liver plays a negligible part in the lactate and pyruvate changes in shock.

F. ENGEL *et alii* (*The Journal of Experimental Medicine*, January, 1944) have continued the investigation of Russell *et alii* with reference to the role of the liver and the hepatic circulation in the metabolic changes during hæmorrhagic shock. In a series of rats subjected to hæmorrhage and shock a high negative correlation was found between the portal and peripheral venous oxygen saturations and the arterial blood pressure on the one hand and the blood amino nitrogen levels on the other, and a high positive correlation between the portal and peripheral oxygen saturations and between each of these and the blood pressure. In five cats subjected to hæmorrhage and shock the rise in the plasma amino nitrogen content and the fall in the peripheral and portal venous oxygen saturations were confirmed. Further, it was shown that the hepatic vein oxygen saturation falls early in shock, while the arterial oxygen saturation showed no alteration except terminally, when it may fall also. Ligation of the hepatic artery in rats did not affect the liver's ability to deaminate amino acids. Hæmorrhage in a series of rats whose hepatic arteries were ligated did not produce any greater rise in the blood amino nitrogen than a similar hæmorrhage in normal rats. The hepatic artery probably cannot compensate to any degree for the decrease in portal blood flow in shock. An operation was devised whereby the viscera and portal circulation of the rat were eliminated and the liver maintained only on its arterial circulation. The ability of such a liver to metabolize amino acids was found to be less than that of either the normal liver or of the liver whose hepatic artery was ligated, and to have very little reserve. On complete occlusion of the circulation to the rat liver this organ was found to resist anoxia up to fifteen minutes. With further anoxia irreversible damage to this organ's ability to handle amino acids occurred. It is concluded that the rise of blood amino nitrogen content during shock results from an increased breakdown of protein in the peripheral tissues, the products of which accumulate, either because they do not circulate through the liver at a sufficiently rapid rate, or because with continued anoxia intrinsic damage may occur to the hepatic parenchyma so that it cannot dispose of amino acids.

Kidney Metabolism.

S. B. RASKA (*The Journal of Experimental Medicine*, July, 1943) has investigated the consumption of oxygen by slices of kidney tissue of dogs made hypertensive by the Goldblatt technique. The respiration of the ischæmic kidney tissue was found to be much less than that of the normal kidney. Further, a marked reduction in oxidizing ability, as measured by the oxygen uptake and ammonia formation in the presence of the added amines and amino acids, tyramine, isoamylamine, *dl*-alanine, and *l*-aspartic acid, was observed. Extracts of the kidneys were made and tested for amine oxidase, amino acid oxidase, and polyphenol oxidase activity by measuring the increased oxygen consumption and ammonia formation in the presence of the substrates listed above with the addition of *l*-epinephrine, histamine and *dl*- and *l*-dihydroxyphenylalanine. The preparations from ischæmic kidneys of dogs and rabbits showed much lower activity. Animals with varying degrees of constriction of the renal arteries and therefore varying degrees of renal ischæmia were prepared and studied. The results with these animals suggested a direct relationship between the degree of renal ischæmia and the decrease in oxidizing power of the tissue.

MEDICINE.

Chronic Ulcerative Colitis.

C. J. DRUECK (*American Journal of Digestive Diseases*, January, 1944) describes the treatment of chronic ulcerative colitis. Colicky pains, diarrhœa and the passage of mucus or blood may be due to food poisoning or amœbic or bacillary dysentery. Chronic or relapsing states of this kind are indistinguishable from non-specific ulcerative colitis. Treatment consists of rest and psychological adjustment, or hospitalization. Phenobarbital, tincture of belladonna (30 minims daily) or camphorated tincture of opium may be useful. Diet in the diarrhœal stage excludes raw fruit and vegetables, beans and cabbage, but includes meat, fish, milk, bread, butter, eggs, bananas and cooked vegetables. After this an adequate and well-balanced diet is given. Abundant fluids, if necessary saline solution and glucose given by the intravenous method, are needed. Calcium gluconate, ten cubic centimetres of a 10% solution given intravenously, and milk given orally, and bismuth or kaolin, one drachm of either, after each motion, may be helpful. Sulphonamides and iodoxyquinoline sulphonate may be given, the latter in doses of four grains every six hours during the first day, eight grains every six hours during the second day, and twelve grains every six hours for the next seven days, followed by a rest for one week, and then repeated three or four times. In some cases six ounces of a 2% solution of sodium iodoxyquinoline sulphonate were run into the bowel, retained for three to five minutes and run out again, the procedure being repeated until one quart of solution was used. The results of this treatment were good in three cases reported.

XUM

British Medical Association News.

MEDICO-POLITICAL.

THE Victorian Branch of the British Medical Association recently appointed special committees to report on the additions which have been deemed necessary to existing consultant, general-practitioner and hospital services to provide a medical service of optimum efficiency for the people of Australia. Some of these reports have been prepared and will be published from time to time in this journal. The first report on group practice is as follows.

Report of Special Committee on "Group Practice".

1. "Group practice", for the purpose of this report, is defined as: "The practice of medicine conducted by a team of not less than three medical practitioners entirely or almost entirely self-contained as regards their ability to diagnose completely and to treat fully any illness that may come before them."

Such a definition presupposes that each member of a group is trained, or is in the process of being trained, in the wide school of general practice, that each has before him the ideal of perfecting himself as far as possible in general practice with some working knowledge of all the specialties and, at the same time, of acquiring much more advanced and detailed knowledge in at least one of the specialties.

2. Apart from the specialties of radiology and pathology, the formation of groups of highly trained specialists is not considered desirable nor necessary nor to be of advantage to the community or the profession.

3. The establishment and growth of "groups" must be a gradual process for the following reasons: (*a*) A group of three or more doctors suddenly starting practice in a district already medically serviced would over-supply the district and create over-competition and discord in the profession. (*b*) The capital cost of establishing group practice is heavy. (*c*) Any attempt to start group practices suddenly "in full blast" on a large scale might easily fail, as most doctors have had no experience of that type of practice.

4. A group of three may successfully produce a combined practice on a partnership basis, but it is considered that preferably, and particularly in larger groups, there should be a senior man regarded as the "managing director" and responsible for the maintenance of standards and discipline within the group.

5. In addition to its senior members, each group should recruit juniors: (*a*) To bring new ideas and fresh energy to the group. (*b*) To do the minor work of the group. (*c*) To be trained in general practice and gradually in special practice. (*d*) Ultimately to succeed the seniors.

6. Basically a group should consist of general practitioners with specialization of its members to a greater or lesser degree in some or all of the following: (*a*) medicine; (*b*) surgery; (*c*) pædiatrics; (*d*) obstetrics and/or gynæcology; (*e*) dermatology; (*f*) ophthalmology and/or oto-rhino-laryngology; (*g*) pathology; (*h*) radiology.

Pathological and radiological services within a group should be limited to those which can be provided by a competent technician working in collaboration with one or more members of the group possessing pathological or radiological knowledge, but special investigations should be delegated to pathologists and radiologists in specialist practice.

7. The equipment of a group should include a building sufficiently large and convenient to enable the members to carry out their work as expeditiously as possible and without getting in each other's way, with accommodation for clerical and nursing staff and necessary apparatus.

Adequate and convenient hospital facilities are essential, and, in fact, group practice might well, in many instances, be centred on existing hospitals.

8. Group practice will succeed only in so far that it gives better service to the community, advantages to the doctors themselves being secondary.

To the community it offers an improved standard of medical service, but whether that service can be provided at any lower cost is questionable. In a group more diagnostic measures will be undertaken with possibly increased expense to the patient, but much work done within the confines of the group will cost less than it would if the patient were referred to several independent specialists. It must be remembered that the inherent costs of efficient modern medical practice are high and it would be a mistake and almost certainly misleading to advocate group practice as a means of lowering medical costs.

9. To the medical profession group practice offers: (*a*) Constant close association of colleagues in the daily work with the stimulus of their cooperation and criticism. (*b*) A system of apprenticeship especially valuable to junior men. (*c*) The opportunity to specialize while carrying on general work and the family connexion with patients. (*d*) Opportunity for leisure and post-graduate study with, during illness, personal security and continuity of service to patients. (As a corollary facilities for post-graduate training must be improved.) (*e*) Better working conditions with better equipment and more adequate technical assistance with removal of the venue of practice from the doctor's home.

Disadvantages to the profession are: (*a*) The cost of establishing and maintaining a group practice. (*b*) The difficulty of establishing and holding together a congenial group of doctors.

10. Group practice is obviously impossible in one man towns and possible to a limited extent only in two or three man towns, but in larger towns and suburbs the formation of groups should be encouraged on the widest possible basis to take in all non-specialist practising doctors, and not merely to substitute rivalry and competition between groups for the rivalry and competition of doctors practising as individuals.

11. No attempt has been made in this report to indicate how the facilities offered by group practice may be brought within reach of all persons in the community.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

THE thirty-eighth annual meeting of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on March 8, 1944, DR. ALAN MCCUTCHEON, the President, in the chair. Part of this report was published in the issue of May 20, 1944.

Bilateral Pulmonary Hydatid Cysts.

DR. WILFRED FORSTER said that his patient, who had suffered from bilateral pulmonary hydatid cysts, had died soon after an operation on the right side. The child commenced to walk at the age of fourteen months, and soon the parents noticed a limp. After a radiological examination at the age of three years, she was admitted to hospital for reduction of bilateral congenital dislocation of the hips. A series of manipulations and applications of plaster casts followed, and finally she was transferred to Frankston. The dislocation of the right hip was reduced fairly easily, but that of the left hip was impossible to reduce because of adduction spasm and anterior rotation of the neck of the femur. Dr. Forster said that he performed osteotomy and rotation of the shaft of the femur on the neck. The child developed a cough with fever, and to everyone's surprise X-ray examination revealed a hydatid cyst in the upper part of each lung, that in the right lung being larger and situated more anteriorly, whilst that in the left lung was slightly smaller and situated posteriorly. The Casoni skin test for hydatid disease produced negative results, as also did the complement fixation test.

Dr. Forster went on to say that it was decided to attack the cyst in the right lung first. When the incision was made, it looked a simple matter to remove the hydatid cyst. He did this after evacuating the contents. The wound was closed without drainage, the operation taking in all about half an hour. The child appeared quite well for a day or two. Then subcutaneous emphysema developed over the neck and the right side of the chest. Her condition became worse. X-ray examination revealed a right pneumothorax with collapse of the right lung. Aspiration of the chest soon revealed that infection had occurred with the production of a pyopneumothorax. Death followed twenty days after the operation. At autopsy, Dr. Reginald Webster had verified the presence of pyopneumothorax. The cyst in the left lung was intact. The lungs were retained as museum specimens, and Dr. Forster displayed the mounted specimens. Dr. Webster was unable to demonstrate hydatid infestation elsewhere but in the lung.

Dr. Forster said that the child was small and underdeveloped, and as she had been in a plaster cast for many months, she was not a "good risk". However, possibly the mistake was made in not packing the chest and promoting adhesions to prevent pneumothorax, or perhaps it was incorrect not to put in a drainage tube. At operation it

had looked a simple task to remove the hydatid cyst, and these measures did not suggest themselves. Dr. Forster said that he had never seen such extensive emphysema develop as in this case. The case was discussed to demonstrate one of the pitfalls in the surgery of hydatid disease.

Complications of Lung Hydatid Cysts.

DR. J. G. WHITAKER said that he intended to show five patients suffering from pulmonary hydatid cysts, to demonstrate various complications which might occur. Most of these patients were under the care of Dr. J. W. Grieve, and they were shown by his courtesy. None of the cases reflected credit on the surgical treatment. Operation upon pulmonary hydatid cysts was complicated, because this was not the complete picture. Hydatid infestation was a widespread, blood-borne infestation, and in children especially, hydatid cysts frequently existed elsewhere than in the lung.

Dr. Whitaker's first patient was a girl, aged four years and ten months. She had had a cough since an attack of pertussis three months previously. The sputum was yellow in colour, not copious or blood-stained. For the last three weeks she had had pain aggravated by breathing over the left side of the chest. She had been losing weight. On examination, she was a pale, undernourished little girl. Movement was poor over the left side of the chest, and the percussion note was dull over the left upper quadrant of the chest anteriorly. The breath sounds were diminished over this area, and a pleural friction rub was audible. The Mantoux skin test produced a negative result with tuberculin diluted one in 1,000 and one in 100. The sputum contained pneumococci, but no tubercle bacilli. X-ray examination revealed consolidation of the left lung. A diagnosis of left lobar pneumonia was made. The child's condition improved sufficiently for her to be discharged from hospital, but she was readmitted two months later with a history of hæmoptysis and the production of copious sputum for six days. The signs in the left side of the chest were much the same as before. Aspiration was performed and thirty cubic centimetres of clear fluid were obtained. The fluid was examined for hydatid hooklets and scolices without success. An X-ray examination at this time revealed hydropneumothorax. The Casoni skin test produced a negative result, but the hydatid complement fixation test returned a strongly positive result. A blood examination showed that the leucocytes numbered 21,000 per cubic millimetre, and the differential leucocyte cell count showed that 39% were polymorphonuclear cells, 52% were lymphocytes, 7% were monocytes and 2% were eosinophile cells. X-ray examination of the abdomen revealed what appeared to be a hydatid cyst in the liver. The child again recovered and was sent to a convalescent hospital. She was readmitted to hospital three months later. Again the signs in the left side of the chest were as before, and in addition tubular breath sounds and crepitations at the apex of the left lung were present. The temperature was swinging between 99° F. and 101° F. Her condition improved after a course of sulphapyridine. After another three months there was another recurrence of pneumonia. Again the acute attack was weathered, and the last radiograph revealed some clearing in the left lung shadows. No surgical procedures were instituted. The case demonstrated hydatid disease complicated by recurring pneumonia.

Dr. Whitaker's second patient was a girl, aged thirteen years and five months. Her illness had begun two years earlier, when she had recurrent attacks of pain in the right hypochondrium and right loin. These attacks lasted two or three days and were associated with fever and anorexia. Twenty months prior to the meeting, she had right-sided pneumonia, and after this attacks of pain became more frequent. She had never been jaundiced and had no cough. On examination, her temperature was 100° F. The breath sounds were diminished and the percussion note was dull over the base of the right lung. Examination of the abdomen revealed a large mass in the right hypochondrium. The mass was about five inches in diameter. It was not tender. Its surface was irregular, and it was ballottable from the flank. An X-ray examination was made. The right half of the diaphragm was much elevated. Consolidation was present in the lower half of the right lung field. The appearance suggested hydatid cysts below the right half of the diaphragm, in the right lower lobe of the liver and in the anterior aspect of the lower lobe of the right lung. Both the Casoni test and the complement fixation test for hydatid disease produced strongly positive results. The Wassermann test failed to produce a reaction. At the first operation, two hydatid cysts were removed from the right lobe of the liver. Convalescence was satisfactory, and the patient was discharged from hospital ten days after operation. She remained well and attended the out-patient department, where she was radiologically examined at intervals. An X-ray picture was taken two months after the operation. The appearances suggested a cyst in the anterior aspect of the middle third of the right lung. It was hoped that spontaneous rupture and healing would take place.

Ten months after the first operation, the chest was opened under nitrous oxide and oxygen anæsthesia. Portion of the seventh rib was removed posteriorly. The cyst could not be visualized. No clue was obtained by compressing the lung in all directions. Aspiration was not performed. The wound was closed with drainage. After the operation, a pleural effusion developed. Aspiration of the thoracic cavity recovered 300 cubic centimetres of turbid, bile-coloured fluid. On pathological examination, the presence of bile was verified, but no pus cells, scolices, hooklets or fragments of laminated membrane were demonstrable. Since then aspiration had been performed on three occasions. On the last occasion 875 cubic centimetres of fluid were removed. The last X-ray examination revealed that the right half of the diaphragm was much raised; adhesions were pulling it upwards, but it was thought that a subphrenic hydatid cyst might be pushing it from below. The pulmonary cyst in the middle third of the lung and projecting from the hilus appeared to be still present. Considerable pleural thickening extended over the upper half of the right lung.

Dr. Whitaker said that this case demonstrated multiple hydatid cysts, the difficulty of approach to a deep-seated hydatid cyst, and rupture through the diaphragm of a subphrenic hydatid cyst with the production of a bile-stained pleural effusion. Dr. Whitaker said that it was the first time he had experienced this complication. The child was never jaundiced. Bile in the peritoneum apparently was more prone to produce the icteric state. Dr. Whitaker concluded by emphasizing the value of a transverse incision in upper abdominal work, especially if a large exposure was required.

Dr. Whitaker's third patient was a boy, aged nine years and five months. The child had lived in the country until four years prior to the meeting. For the last three or four years he had complained of an occasional pain in the left lower quadrant of the chest; this pain was occasionally worse with breathing. Three weeks prior to his admission to hospital the pain appeared more severe, and he became more feverish. He was admitted to hospital on January 25, 1944. Examination revealed him to be a pale, thin boy. Movement was diminished over the left side of the chest. The percussion note was hyperresonant at the base of the left lung anteriorly. The breath sounds were diminished at the base of the left lung anteriorly. Tubular breath sounds were audible at the base of the left lung posteriorly. An X-ray examination was made. The appearances suggested a hydatid cyst at the base of the left lung posteriorly; there was no evidence of a hydatid cyst in the liver. The hydatid complement fixation test produced a negative result. The Casoni test produced a positive result after twelve hours. The Mantoux test failed to produce a reaction.

Dr. Whitaker said that in this case operative treatment was held in abeyance; the hydatid cyst was very small in size, and he was conservative in such cases. He recalled an occasion on which rupture of such a hydatid cyst occurred during exploration, with the production of an acute anaphylactic shock from which the patient nearly died. There was, however, a difference of opinion in regard to these small hydatid cysts, and it was a fine point to decide. He asked for an expression of opinion in this particular instance. Dr. Whitaker reminded the audience that the vast majority of hydatid cysts were never seen by the medical profession.

Dr. Whitaker's fourth patient was a boy, aged six years and six months. The child had always lived in the country. Five weeks prior to his admission to hospital he complained of nausea and vague abdominal pains. On examination, he appeared a healthy child. The percussion note was dull, and the breath sounds were diminished over the right side of the chest. The heart was slightly displaced to the left. The liver was enlarged two fingers' breadth below the right costal margin. No tumour was felt. The Casoni test produced a positive reaction. X-ray examination revealed a dense area over the lower two-thirds of the right side of the chest, with a rounded denser area above the medial aspect of the right side of the diaphragm. At the first operation a cyst was removed from the anterior aspect of the right lobe of the liver. Convalescence was uneventful. Five weeks later rib resection was performed and an iodine pack was inserted. One month after this, the wound was repacked with iodine gauze. The intrathoracic cyst was

found to be extrapulmonary and attached to the side wall of the mediastinum. Convalescence from this operation was uneventful. Dr. Whitaker said that a number of hydatid cysts were not clearly demonstrable radiographically. The present case was an example of an extrapulmonary mediastinal hydatid cyst with associated pulmonary changes. The hepatic hydatid cyst was removed as a preliminary, and a two-stage operation was attempted on the intrathoracic cyst.

Dr. Whitaker's fifth patient was a boy, aged four years and six months; he had suffered from pneumonia on five occasions. The boy had lived in the country. He was quite well until the age of two years, when he developed "pneumonia" three times in one year. He had been a patient at a country hospital, where he was radiologically examined. This examination revealed a large round opaque area in the middle of the left lung. On June 23, 1942, a rib resection was performed. The cyst was felt, but was not removed. The child was referred to the Children's Hospital on September 9. He had no cough or sputum.

On examination, he was a healthy looking little boy. General examination revealed no abnormality except a scar on the left side of the chest. The temperature was normal. The Casoni test failed to produce a reaction. The hydatid complement fixation test produced a positive reaction. The Mantoux test failed to produce a reaction with tuberculin diluted 1/1,000. An X-ray examination on September 2, 1942, revealed an area of consolidation spreading from the left hilum; in the lateral view this was revealed as a discrete, rounded area suggestive of a cystic basis. A further X-ray examination on September 10 revealed an area of consolidation containing an inner small ovoid opacity in the lower lobe of the left lung, lateral to and on a level with the left hilum. The patient was discharged to his home on October 22.

He was readmitted to hospital for review on June 6, 1943. He had been very well since his last stay in hospital. On examination, no abnormality was found. The Casoni test still failed to produce a reaction; the hydatid complement fixation test produced a strongly positive reaction. An X-ray examination revealed an area of consolidation larger than in the original films, with a suggestion of cavitation in the centre. The appearances were still not characteristic of hydatid cyst. The point was raised whether tuberculous consolidation or foreign body could be excluded clinically. On June 23 a bronchographic examination was made, but no lipiodol entered the upper lobe of the left lung. He was discharged to his home. The patient was readmitted to hospital on January 1, 1944. He had been well until two weeks before this, when he developed a cough. On examination, the temperature was found to be 101° F. The percussion note was impaired over the apex of the left lung, and the breath sounds were diminished over the same area. No adventitiæ were heard. The temperature quickly returned to normal and the chest condition cleared clinically. The sputum was examined on January 7. No tubercle bacilli were found, but pus cells were present in abundance. An X-ray examination on January 7 revealed bronchopneumonic consolidation in the lower lobe of the right lung and bronchopneumonia in the subapical region of the upper lobe of the left lung. There was a fairly large cavity in the middle third of this lung, with surrounding consolidation. An X-ray examination on February 8 revealed that the area of consolidation in the middle zone of the left lung was less; in the centre of the area of consolidation was a small cavity. On February 17 he was discharged to his home. He was clinically very well.

Dr. Whitaker said that the patient's general condition remained good without operative interference. The left hilar shadow was by no means conclusive evidence of hydatid disease. In view of the positive response to the complement fixation test, however, this was likely, and the case furnished an example of a small cyst that had ruptured with the possibility of spontaneous cure. In conclusion, Dr. Whitaker expressed his thanks to Dr. Noreen Nicholson for collecting and classifying the cases for the purpose of the presentation.

DR. C. J. OFFICER BROWN said that much of the confusion about hydatid disease of the lung was due to the fact that most of the literature on the subject antedated the present era of thoracic surgery. Whilst much was owed to the skill and courage of surgeons in this country and in other regions where the disease occurred for the bold way in which they had tackled this problem without many of the advantages now available, it had to be realized that much of their teaching and practice needed revision. As a result of the developments in radiology, in anæsthesia and in thoracic surgery, the diagnosis and localization of intrathoracic conditions were much more accurate than they used to be, and it was possible to carry out safely operations that could not have been considered a few years earlier.

The patient with hydatid disease of the lung might present himself for treatment in one of three stages. In the first stage he had a simple intact cyst or cysts. The majority of these cysts were clinically latent; but they might cause cough, wheezing, hæmoptysis or pleural pain. Dr. Brown said that he had operated on 31 patients with hydatid cysts of the lung and had removed a total of 33 cysts, and in every case one or more bronchial openings were demonstrated in the wall of the adventitial cavity. He considered it unwise to treat any cyst on the assumption that such a fistula was not present.

The second stage followed rupture of the cyst. In a few cases rupture took place into the pleural cavity, and because of the presence of a bronchial fistula this was always accompanied by some intrabronchial leakage of fluid, and always resulted in a hydatid hydropneumothorax. The fistula might close, and with absorption of the fluid and air the lung might reexpand. The membrane might remain in the lung, or if it had been expelled into the pleural cavity it remained there, and in either case it should be removed. If infection became superimposed, a pyopneumothorax resulted; this might present an extremely difficult problem of treatment. Secondary cysts might arise from scolices spilled into the pleural cavity, when the condition became one of secondary pleural echinococcosis. More commonly, the cyst ruptured into a bronchus and discharged its fluid. This was always a dramatic incident, with much coughing and distress, and occasionally the patient drowned in the fluid, or severe anaphylactic shock might develop. Cases had been reported in which the patient was suffocated by impaction of the membrane in the larynx. In a certain proportion of cases, especially when the cysts were small, the membrane might be evacuated with the fluid and a spontaneous cure result. Usually the membrane remained behind, and although it might ultimately be discharged as a result of bacterial action and the disintegrating effects of tissue ferments, the process was slow and might take months or even years. During this period the membrane acted as an infected foreign body in the lung substance, and was no more harmless than a tooth or other infected foreign body. Atelectasis, fibrosis and bronchiectasis always occurred to some extent, and the cavity containing the membrane might become lined by epithelium so that it could never be obliterated. Incidentally, the lining of such cavities in the lung was normal respiratory epithelium and not squamous epithelium, as was often stated. X-ray examination shortly after rupture of the cyst would show the cavity to be partially filled with air and with a fluid level, and the crumpled membrane could usually be seen above the fluid. Later the fluid was all discharged and the adventitia closed down on the retained membrane, and only a vague opaque area was left to represent the cyst; this might easily be looked on as scar tissue, but was in fact the crumpled membrane retained in the lung. Occasionally daughter cysts might develop in these collapsed cysts, but as a rule the hydatid element died, infection became superimposed and the problem became that of a foreign body in the lung. It was surprising how well the patient usually remained, even when severe suppuration was present around the membrane; this was probably because the pus was contained within a well-defined adventitia, which had freely drained into a bronchus.

The third stage followed the eventual evacuation of the retained membrane, either spontaneously or by operation. Symptoms might persist as a result of the residual lung changes, and Dr. Brown said that he had on two occasions had to perform lobectomy for the relief of these symptoms. One patient developed persistent severe hæmoptysis twenty-five years after spontaneous evacuation of his cyst, and the other sought relief ten years after an eventful surgical cure because of recurrent febrile attacks with hæmoptysis and the expectoration of foul pus. Several other patients whom Dr. Brown had seen in this stage had not had sufficient disability to justify radical treatment, but they were not so well as they might have been. Lobectomy ought never to be considered as the primary treatment of a hydatid cyst.

Dr. Brown then showed the X-ray film of a patient who had suffered from pneumonia and pleurisy fifteen months previously. Six weeks later he had had an hæmoptysis, and this had recurred on and off ever since. He had no energy and could not work. X-ray examination made at the time of the first hæmoptysis revealed a small irregular opaque area at the level of the second left costal cartilage. This had remained unchanged throughout the period of his disability, and Dr. Brown looked on it as a collapsed hydatid

cyst. The Casoni test produced a positive reaction and hydatid complement fixation test no reaction. Subsequent operation revealed the collapsed membrane of a small hydatid cyst, and this was removed.

Dr. Brown said that in his opinion every hydatid cyst that was discovered in the lung should be removed, regardless of its site or its size, because surgical removal was safer and more certain than spontaneous cure. A number of patients whose unsuspected cysts had been discovered by routine X-ray examination for the army or for certain civilian occupations had been operated on successfully and a minimum of time had been lost; but in two cases in which the cyst was recognized in this way and left alone it later ruptured, and both patients went through long, hazardous illnesses before the cysts were finally removed by operation.

Dr. Brown always operated on patients suffering from hydatid cysts of the lung under anæsthesia with ether delivered through a McGill's endotracheal tube with an exhaling valve and a machine which could be used to control endotracheal pressure. A small rubber catheter was passed down the McGill's tube and used to keep the trachea clear of blood, fluid and secretions. The cyst had to be accurately localized by X-ray films taken in two planes, and fluoroscopy was sometimes of value. The cyst would usually be found at least one rib below the level indicated by the standard chest film taken at full inspiration, and unless the surgeon was sure of its localization he should have X-ray films taken at full expiration with the patient in the position he would occupy on the operating table. All cysts came into contact with the surface of the lung at some spot. It might be on the mediastinal or diaphragmatic surface, or in one of the fissures; but because the costal surface was so much greater than all the other surfaces, most cysts made contact somewhere with the chest wall. In most cases it was possible to approach the cyst directly by excising four inches of a rib immediately over it and opening the pleura. If localization was accurate, the pearly appearance of an intact cyst was readily recognized; but if the cyst had collapsed, all that was found was a thickened scar at the point of its approach to the surface of the lung, and a resistance was felt underlying this. Although no fluid would be obtained by aspiration of a collapsed cyst, its presence could be confirmed by the change of resistance to the passage of a needle as soon as it had passed through the adventitia, which usually occurred at a depth of about one-eighth of an inch. Sometimes thick pus exuded through the needle hole. The scarred area could be incised without any bleeding and the membrane readily picked out. When the cyst did not come into contact with the chest wall, a free opening designed to give the most direct approach to it should be made and the place where it reached the lung surface exposed and isolated by packing within the pleural cavity; the cyst was then dealt with as before. The fluid was removed from simple cysts by suction, while the anæsthetist maintained a positive pressure in the bronchial tree to prevent the aspiration of fluid into the lung. After the membrane had been removed and the cavity swabbed out, a de Pezzer type of tube was inserted and the opening in the adventitia was tightly closed around the tube and at the same time sutured to the opening in the chest wall, which was then closed. Sometimes a second tube was inserted in a dependent position to drain the pleural cavity if it was feared that a hæmothorax might result, or if the pleura had been much soiled with pus. This tube must always be attached to a closed drainage bottle, and if the chest cavity was dry in forty-eight hours it should be removed.

Dr. Brown said that most of his patients had been discharged from hospital with the wound healed in from ten to fourteen days, and only two had remained in hospital for more than three weeks. One of these was suffering from secondary pleural echinococcosis, and the other developed an empyema, which was the only complication in the series.

Dr. Brown said that he did not use a two-stage operation because he did not consider that it was necessary, and because he know of no certain way of producing adequate adhesions. It was much more hazardous to rely on inadequate adhesions than to perform a one-stage operation planned to avoid the pitfalls of an open pneumothorax and the spilling of scolices. Multiplicity of cysts had not been common in Dr. Brown's series. Two patients had had associated liver cysts, and one patient had had two and another three cysts in the lungs. All the cysts were removed except one simple lung cyst in a man, aged seventy-three years, from whose other lung a ruptured cyst was removed. Six months later this cyst ruptured, and next day the old man had a stroke and died. After any operation on the chest, attention should be paid to the way in which the patient breathed. Many people, as a result of pain and fixation, developed a habit of unilateral breathing, and unless this was corrected by breathing exercises it might remain a lifelong handicap.

DR. H. DOUGLAS STEPHENS congratulated Dr. Brown on his able exposition of treatment in those cases associated with rupture and collapse. He said that in his experience quite a number of pulmonary hydatid cysts were multiple—in children, at any rate. Very often small cysts were encountered close to the mediastinum. These might rupture, and the rupture might be followed by hæmoptysis with spontaneous recovery. Dr. Stephens wondered whether adults were different in this respect. Such cysts should grow bigger as the patient aged, and should approach the visceral pleura. He asked whether Dr. Brown would advise waiting in these cases, or perform a difficult operation to reach the cysts in the mediastinum.

Dr. Brown said that he had little to comment on this question, as he had had no experience of small cysts in children. He believed that quite a lot of people coughed up the cyst and "got away with it". But these patients had to be watched carefully.

Dr. Stephens asked Dr. Brown how long he would wait in such cases.

Dr. Brown said that he could not answer that question.

Dr. Stephens also asked whether Dr. Brown could say at what time the responses to the Casoni reaction and the complement fixation test became negative.

Dr. Brown replied that the response to the Casoni test never did so, and the response to the complement fixation test was negative after a year or two. It should be remembered that in the case of a patient who had had a cyst operated on, the Casoni test ceased to be of value as a diagnostic test.

DR. ALAN MCCUTCHEON reminded those present of the diagnostic method used twenty-four years earlier by a surgeon in New Zealand. The operator had put his two fingers into the wound and exclaimed: "Hydatid all right, I can feel the hooklets!"

Dr. Whitaker, in reply, said that he was indebted to Dr. Brown for clarifying many points. At the Children's Hospital, multiple lesions existed in a much greater percentage of cases than that experienced by Dr. Brown (two out of thirty-three). Dr. Whitaker said he was perplexed by the child with bile in the pleural cavity. He suspected that she had more hydatid cysts in the liver. Concerning anæsthesia, Dr. Brown's method sounded a very useful one. Dr. Whitaker had not had the opportunity of seeing Dr. Brown at work with this form of anæsthesia. Dr. Whitaker said that at the Children's Hospital he had managed satisfactorily with nitrous oxide given by the "closed" method, and "Avertin". But with a closed pneumothorax, one would want a more efficient system than a closed nitrous oxide bag. Dr. Whitaker said that he was glad to hear that Dr. Brown insisted on the necessity for drainage. This seemed to be an important part of the procedure.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 93, of May 11, 1944.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointments.—Ronald Munro Ford and John Hamilton Stace are appointed Surgeon Lieutenants, dated 10th March, 1944.

Promotion.—Surgeon Lieutenant Edward Eric Keith Bottomley is promoted to the rank of Acting Surgeon Lieutenant-Commander, dated 31st March, 1944.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The following Flight Lieutenants are confirmed in their probationary appointments with effect from the dates indicated: B. Starr (257522), M. Barry (267236), C. S. Kerr (285947), 2nd February, 1944, W. M. Davies (256407), 15th February, 1944, J. Tyrer (267564), 28th February, 1944.

Squadron Leader J. B. G. Gibson (277428) is transferred to part-time duty with effect from 16th October, 1943.

The following officers are transferred from the Royal Australian Air Force Medical Reserve to the Active List for full-time duty with effect from the dates indicated: Wing Commander N. E. H. Box (1172) (part-time), 8th February, 1944, Squadron Leader H. M. Webber (261893), 6th March, 1944.

Gordon William Browne, M.B., B.S. (267689) is appointed to a commission on probation with the rank of Flight Lieutenant for full-time duty with effect from 13th March, 1944.

The appointment of Flight Lieutenant M. Ross (257166) is terminated at her own request with effect from 29th March, 1944.

The appointment of Flight Lieutenant M. J. M. Black (265944) is terminated on medical grounds with effect from 31st March, 1944.

Reserve: Medical Branch.

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Edward Joseph Khan, M.B., B.S. (277495), Robert Alfred Russell, M.B., B.S. (287441), Russell Markham O'Donohue, M.B. (267672), 14th February, 1944, Charles Francis McCann, M.B., B.S. (257631), 26th February, 1944.—(Ex. Min. No. 129—Approved 10th May, 1944.)

Squadron Leader A. C. Blumer (261469) is transferred from the Active List with effect from 19th April, 1944.—(Ex. Min. No. 132—Approved 10th May, 1944.)

CASUALTIES.

ACCORDING to the casualty list received on May 18, 1944, Captain M. A. Gardner, A.A.M.C., Ryde, has been removed from the "seriously ill" list.

Notice.

THE Mental Hospitals Department of New South Wales, in conjunction with the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association, will hold a clinical meeting at Broughton Hall Psychiatric Clinic, Leichhardt, on Thursday, June 15, 1944, at 8 o'clock p.m. All members of the medical profession are invited to be present.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Greenberg, Harris Phillip, M.B., B.S., 1944 (Univ. Sydney), St. George District Hospital, Kogarah.

Thwaite, Thomas John, M.B., B.S., 1944 (Univ. Sydney), B.Sc., 1942, Royal Prince Alfred Hospital, Camperdown.

Hornbrook, John Wallis, M.B., B.S., 1944 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Robertson, John Arnold Struan, M.B., B.S., 1943 (Univ. Sydney), Sydney Hospital, Sydney.

Hill (Bulteau), Marie Eleanor, M.B., B.S., 1944 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Furber, Thomas Rupert Maynard, M.B., B.S., 1944 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Furber, John Frederick Maynard, M.B., B.S., 1944 (Univ. Sydney), 9, Wunulla Road, Point Piper.

Obituary.

JOHN GRANTLEY VINCENT SHELTON.

WE regret to announce the death of Dr. John Grantley Vincent Shelton, which occurred on May 16, 1944, at Malvern, Victoria.

Diary for the Month.

MAY 30.—Federal Council, B.M.A.: Meeting in Sydney.
JUNE 1.—New South Wales Branch, B.M.A.: Special Groups Committee.
JUNE 2.—Queensland Branch, B.M.A.: Branch Meeting (Joseph Bancroft Memorial Lecture).
JUNE 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 7.—Victorian Branch, B.M.A.: Branch Meeting.
JUNE 7.—Western Australian Branch, B.M.A.: Council Meeting.
JUNE 9.—Queensland Branch, B.M.A.: Council Meeting.
JUNE 9.—Victorian Branch, B.M.A.: Legislative Subcommittee.
JUNE 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 13.—Tasmanian Branch, B.M.A.: Branch Meeting.
JUNE 16.—Victorian Branch, B.M.A.: Ethics Subcommittee.
JUNE 19.—Victorian Branch, B.M.A.: Hospital Subcommittee.
JUNE 19.—Victorian Branch, B.M.A.: Finance Subcommittee.
JUNE 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 20.—Victorian Branch, B.M.A.: Organization Subcommittee.
JUNE 21.—Western Australian Branch, B.M.A.: Branch Meeting.
JUNE 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 22.—Victorian Branch, B.M.A.: Executive Meeting.
JUNE 23.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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